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SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS ASSOCIATED WITH ANTHRACOSIS AND SILICOSIS IN COAL MINERS

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TUBERCULOSIS associated with anthracosis and silicosis is a grave disease. Sanatorium treatment gave good results in about 15 per cent of patients,[1] pneumothorax[2] and surgical collapse[3, 4] were rarely successful. The appearance of antibiotic therapy brought little change in the problem although the use of associated antibiotics led to cure in about 25 per cent of patients so treated.[6, 7, 8]

The gravity of the situation induced surgeons to try resection of these lesions, though with some hesitation in sclerous, rigid, and infiltrated lungs.

The first resections were pneumonectomies, performed after a diagnosis of cancer,[9, 10] followed in 1952 by operations after a diagnosis of silicotuberculosis.[11] The cases were scarce for years,[12, 13] and in 1956 the first experiences in 31 patients were published in France.[14] Soon results of the method appeared in different countries—Belgium, Czechoslovakia, France, and the United States.[15-18] Special aspects, such as the treatment of massive hemoptysis, were studied.[19]

MATERIAL

We now have treated 159 patients. All these patients were tuberculous and had, at the moment of their operation or before, positive sputum. In 2 patients, tuberculosis was proved only by histologic studies. All the patients had chest x-ray studies which proved a coniotic background. We eliminated from this study coal miners with apparently normal backgrounds although even

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these patients behave abnormally with the use of antibiotics. We used the radiologic classification as proposed by the silicosis centers of Cardiff (England) and Douai (France).

PATHOLOGY

There is a difference between cases in which tuberculous follicles exist in the neighborhood of silicotic lesions and cases in which there is an intimate association of both processes to form a new disease: silicotuberculosis. In addition to silicosis[20] another form of coniosis has been isolated–the "coal-miners disease" or anthracosis. This form has been found in Belgium, Japan, France, and U.S.S.R. It becomes more frequent as means of protection against silicosis increase, and brought us to a classification derived from radiologic, surgical, and histologic studies.

1. *Tuberculosis on a background of anthracosis* is an ordinary form of tuberculosis (cavities, nodules, and follicles) on a coniotic background.[21, 22, 23] Fibroconiotic nodules with perifocal emphysema also occur (Fig. 1).

2. *Anthracotuberculosis* takes the aspect of solid, marble-like masses or thick-walled caverns, in contact with dense, black, and nonaerated tissue. There is "dust" in the necrotic tissue and the tuberculous follicles are often modified. There are fibroconiotic nodules in all the lung but no hyaline tissue (Fig. 2).

3. *Silicotuberculosis.* (a) In silicotuberculosis with a nodular background the tuberculous lesions lie among typical sclerohyaline silicotic nodules. In places, the lung is not entirely silicotic, but anthracotic. (b) Excavated pseudotumoral masses occur which offer a surgical problem only in the cases in which the tuberculous process remains well limited (Fig. 3).

Histologic studies of 144 of our 159 patients disclose the following distribution: (a) ordinary tuberculosis plus anthracosis, 37; (b) anthracotuberculosis, 39; and (c) silicotuberculosis—45 silicotic nodules, and 23 silicotic pseudotumors.

RADIOLOGY

There is no exact relationship between the nature of the lesions and their radiologic aspect (Fig. 4). Practically, it can be said that in coal miners of Northern France a punctiform or micronodular background is more sharp and more uniform in silicotuberculosis than in anthracotuberculosis, or tuberculosis associated with anthracosis, whatever is the aspect of the principal lesion, whether cavern or dense excavated lesion. Isolated excavated masses are an expression of anthracotuberculosis. Large nodules and pseudotumors nearly always mean silicotuberculosis. In all cases it is essential to be certain of the localized character of the tuberculous lesions. Bronchoscopy and tomograms must be used to determine this.

CONSIDERATIONS FOR OPERATION

I. *Indications for Operations.*—A resection must not be undertaken until after 6 months of medical treatment. The triple antibiotic association is used.

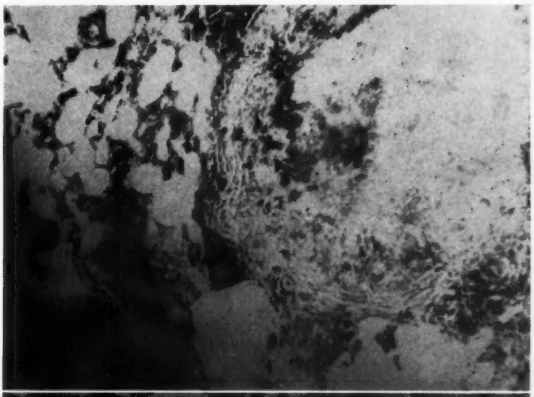

Fig. 1.

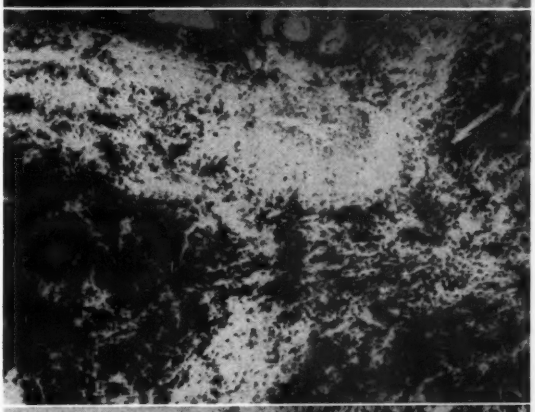

Fig. 2.

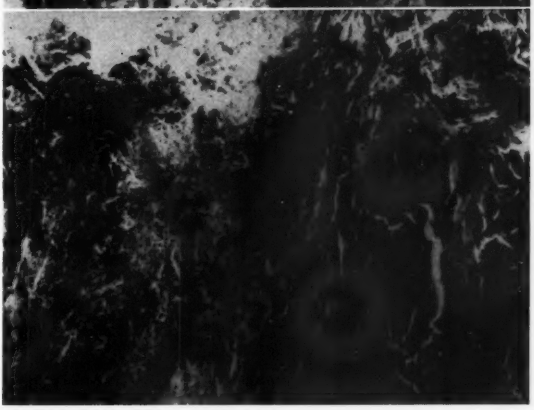

Fig. 3.

Fig. 1.—In the upper right corner of the photomicrograph is seen a typical tuberculous nodule close to a fibroconiotic formation in the left lower part of the slide.

Fig. 2.—Anthracotuberculosis—tuberculous and coniotic lesions are intermingled.

Fig. 3.—Silicotuberculosis—an excavation *(upper left)* is eroding sclerohyaline silicotic tissue.

All patients must be given the opportunity to be cured medically but the time for surgery must not be overlooked when resection becomes necessary. After a period of 6 months, the first re-evaluation may be made; if the lesions appear to be improved, it is wise to continue medical treatment. If the lesions are stationary, and the sputum is negative, the patient must not be considered as cured and surgical treatment is recommended. If the lesions have become more extensive, resection is the only possible solution.

There are factors which influence early surgical treatment: attempts by the patient to escape medical treatment discipline, or the appearance of some degree of resistance or intolerance to antibiotics. (This resistance is almost always increased in coal miners, even with x-ray studies showing very little or no coniotic change.) There also are factors which demand a longer medical preparation, such as old tuberculous scars beyond the territory to be resected.

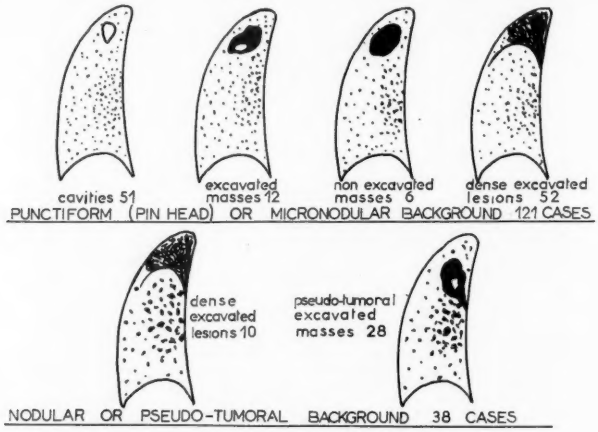
cavities 51
excavated masses 12
non excavated masses 6
dense excavated lesions 52
PUNCTIFORM (PIN HEAD) OR MICRONODULAR BACKGROUND 121 CASES
dense excavated lesions 10
pseudo-tumoral excavated masses 28
NODULAR OR PSEUDO-TUMORAL BACKGROUND 38 CASES

Fig. 4.—Drawing of the x-ray aspects of the lesions (159 cases).

II. *Preoperative Evaluation.*—The patient and the evolution of his disease are studied with respect to age, history, general appearance, weight, temperature, and sputum, among other factors. Nothing can replace a thorough clinical examination. Functional lung and heart studies are important. The shape, localization, and extension of the tuberculous lesion must be accurately known. Its topography must be outlined with precision. The pneumoconiotic environment is determined. If the sputum is positive, a study of the strain must be performed.

The surgical prognosis is excellent for localized tuberculous lesions on a fine pneumoconiotic background (Figs. 5 and 6). However, results are better for anthracotuberculosis than for silicotuberculosis.

Pseudotumoral forms (Fig. 7) may be resected when the tuberculous lesions are strictly unilateral; this demands very accurate x-ray and bronchoscopic studies.

In lesions spreading beyond the limits of a lobe, a segmentectomy may be associated with lobectomy when the extension of the lesion is perfectly

Fig. 5.

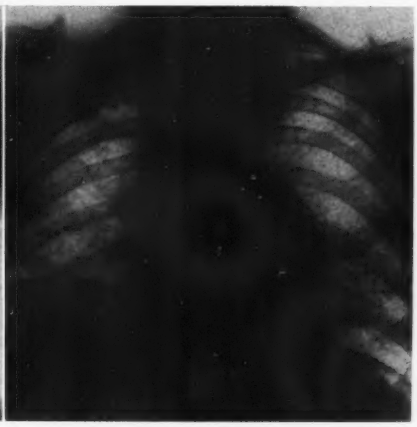

Fig. 6.

Fig. 5.—Silicotuberculosis on a micronodular background.
Fig. 6.—Same patient shown in Fig. 5 after a right upper lobectomy.

Fig. 7.—Pseudotumoral form of silicotuberculosis.

determined before the operation is undertaken. Two of our patients underwent bilateral operations for limited lesions. In old tuberculosis and in patients showing resistance to one or two antibiotics, the operation may be performed under the cover of currently used drugs such as alpha-ethyl-thioisonicotinamide (1314 T.H.)

In summary, the choice indication is a recent, well-limited tuberculosis, well treated by antibiotics for 6 months, in a patient under 50 years of age, showing the punctiform (pin-head) or micronodular form of pneumoconiosis. We reject patients over 55 years of age who are in poor cardiorenal state, patients with diffuse forms of silicotuberculosis, and patients with vital capacities below 2,500 ml. or maximum expiratory volume per second below 1,500 ml. Patients with cor pulmonale are not operated upon.

OPERATION

The anesthetist must avoid carbon dioxide retention within these inelastic lungs. We use posterolateral thoracotomy. Thick pleural adhesions may require a costal resection.

I. *Aspect of the lesions.* The lung is dense and sclerotic. The pleura is thick and pleural adhesions are frequent. The fissures are rarely open. The hilum is fibrous, filled with lymph nodes which hide the bronchi and vessels. The mediastinum is adherent to the lung (especially the phrenic nerve and the azygos vein, which must be avoided).

Lymph nodes and fibrosis often make dissection of vessels and bronchi difficult. Some surgeons advise peripheral dissection and ligature.[18] We prefer to go to the very hilum, which is possible after a cautious, accurate, and sometimes tedious dissection.

II. *The resections* performed were: 107 upper lobectomies, 21 lower lobectomies, 4 bilobectomies, 12 lobectomies plus segmentectomies, 6 segmentectomies, and 9 pneumonectomies, 3 of which were secondary to a former lobectomy.

III. *Operative complications* were represented by vascular wounds in 9 cases without fatal outcome, and a main bronchus wound which required a pneumonectomy. Difficulties and accidents are more common in silicotuberculosis than in anthracotuberculosis.

IV. *The rehabilitation of the thoracic cavity* requires excellent postoperative drainage. These lungs have a greater capacity for expansion than anticipated. It is possible to help it by a temporary pneumoperitoneum. We performed a secondary thoracoplasty on 10 patients only.

POSTOPERATIVE PERIOD

The first 6 months are most important for the future of the patient. The following postoperative complications were encountered.

Hemothorax is not uncommon in those patients with pleural adhesions, greater hilar vascularization, and diminution of lung elasticity (3 deaths).

Wet lung and atelectasis are frequent in miners with chronic bronchitis and also in the older patients who have lost the habit of deep respiration.

Pleural effusions are rare. Residual pneumothorax usually disappears within a few weeks. Pleural infection is exceptional and is treated by thoracoplasty.

Bronchial fistula appeared in 11 cases. By means of an endoscope, we routinely observe the aspect of the suture during the third week. If it does not seem to heal well, we cauterize it with liquid silver nitrate. With this procedure we were able to close 6 bronchial fistulas. For the remaining 5, we performed thoracoplasties. (We use this endoscopic survey and nitratage for all our cases—apart from coniosis—and find it valuable.)

Tuberculous spread occurred in 6 cases.

Generally speaking, the operative risk is greater in patients over 40 years of age (5 deaths in 60 patients over 40 years as against 3 deaths for 99 patients under 40 years). Old lesions, also, which were irregularly treated for a long period of time, offer the greatest risk of complications.

The punctiform (pin-head) and micronodular lungs are less subject to complications than nodular and pseudotumoral lungs. Silicosis is more serious in this respect than anthracosis.

Lobectomy is the best type of resection (128 cases, 4 deaths). Lobectomy plus segmentectomy are a source of complication (16 cases, 2 deaths). Segmentectomy is indicated in very small and well-limited lesions (9 cases, 1 death). Pneumonectomy is rarely indicated (6 cases, 1 death).

Eight patients died during the postoperative period (first 6 months). We had no deaths in the last 40 patients. This mortality rate (8 in 159) is superior to the rate admitted for simple tuberculosis. It is acceptable in regard to the usual evolution of tuberculosis in pneumoconiotic patients. It will decrease if we have the opportunity of operating on patients with recent disease, well treated for 6 months, with good postoperative treatment in sanatorium or hospital.

RESULTS

Of our 159 patients, 141 were operated upon more than 6 months ago and, of these, 133 survive. They have been regularly followed by routine clinical examination, sputum culture, and x-ray studies. Twelve of our patients remained tuberculous or had a new tuberculous lesion, 3 of them with a bronchial fistula (3 deaths).

We never observed an extension of anthracosis, but 3 of our patients had a spread of silicosis. In 9 patients a slight modification of the x-ray picture cannot be attributed with certainty either to an extension of silicosis or of tuberculosis. We do not yet consider them as definitely cured (Table I).

TABLE I

	NUMBER OF RESECTIONS	CURED	UNCURED	UNCERTAIN	DEAD	RETURNED TO LIGHT WORK
6 months—2 years	55	46	3	3	3	
2 years —3 years	44	31	7	4	2	34 out of 78,
3 years —4 years	25	21	3	1		more than 2
4 years —5 years	5	4		1		years after
Beyond 5 years	4	4				operation

After 2 years, complications are rare and the patient may be considered as cured and allowed to return to light work. Failures are to be expected in old tuberculous lesions (more than 3 years of age); in extensive lesions (more than one lobe); in nodular and pseudotumoral coniotic backgrounds; and in patients with positive sputum (11 failures in 84 positive cases, 2 failures in 49 negative cases). Definite cures have been obtained in 65 per cent of the patients, proved clinically by bacteriologic and x-ray studies (Figs. 8-12). This percentage should increase in the future with a better knowledge of this particular surgery.

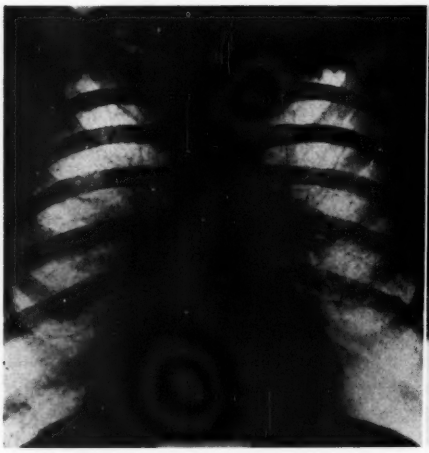

Fig. 8.

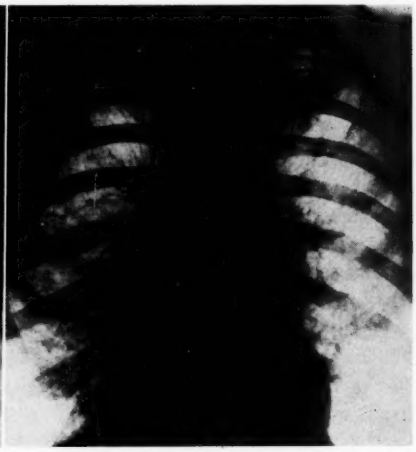

Fig. 9.

Fig. 8.—Limited lesion of the left apex, on a punctiform (pin-head) background.
Fig. 9.—Same patient shown in Fig. 8 after apicodorsal segmentectomy.

Functional results show an important reduction, about 20 to 25 per cent during the first 3 months. We are now reducing it by a better quality of treatment during the immediate postoperative period, insisting on respiratory exercises, and by the frequent institution of a temporary pneumoperitoneum to avoid emphysema of the lung bases.

DISCUSSION AND SUMMARY

Lung resection as a treatment of anthracotuberculosis and silicotuberculosis gives good results under certain conditions.

1. The patients must be prepared by an intense and sufficient medical treatment, which should not be pursued indefinitely.

2. The tuberculous lesion must be well limited in order to be submitted to a well-planned resection.

3. The patient must be closely followed for 2 years following the operation. Failure to cure is more frequent than in ordinary tuberculosis, but a calculated

risk should be taken in these cases, keeping in mind the insufficiency of antibiotic therapy and the usually grave evolution of the disease.

4. Very few postoperative coniotic extensions were observed.

5. Postoperative cardiorespiratory insufficiency never occurred.

6. This new operation will be subject to improvement and changes. It is hoped that a larger experience in this field will standardize it.

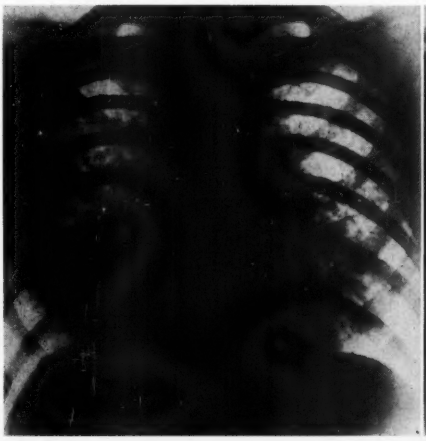

Fig. 10.

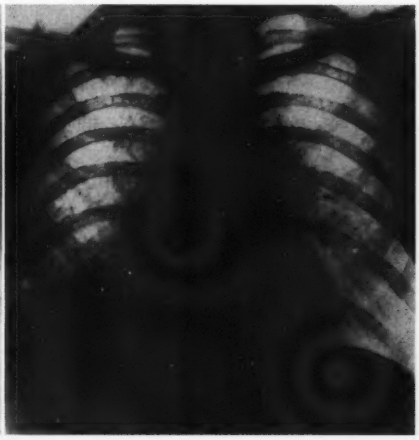

Fig. 11.

Fig. 10.—Bilateral lesion of the right lower lobe and left apicobasal segment.
Fig. 11.—Same patient shown in Fig. 10 after first stage—right lower lobectomy.

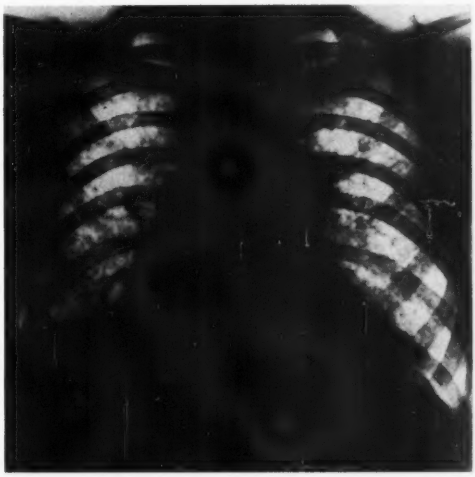

Fig. 12.—Same patient shown in Fig. 10 after second stage—left apicobasal segmentectomy.

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PARADOXICAL RESPIRATION AND "PENDELLUFT"

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PARADOXICAL motion of the chest wall is regularly observed following thoracoplasty and certain types of crushing injury to the chest. The respiratory distress associated with paradoxical respiration is customarily attributed to *pendelluft*, the pendulum-like movement of air from one lung to the other. Despite the fact that descriptions and diagrams of *pendelluft* appear in most textbooks of general and thoracic surgery, there has been no clinical or experimental evidence presented to prove that this phenomenon actually exists. The purpose of the present study is to investigate, from both the theoretical and experimental points of view, the phenomenon of *pendelluft*.

The development of the concept of paradoxical respiration and *pendelluft* parallels the history of extrapleural thoracoplasty. In his early experience with thoracoplasty, Brauer[1] noted that the decostalized portion of the chest wall moved in a direction opposite to that of the remainder of the thorax. With each inspiration, as the chest expanded, the area of operation appeared to sink inward. With each expiration, as the chest collapsed, the paradoxing area appeared to expand. The respiratory distress occurring in patients with paradoxical respiration was attributed to the pendulum-like motion of respiratory gases from one lung to the other. It was postulated that with each inspiration, respiratory gases passed from the lung on the paradoxing side (which appeared to be collapsing) into the contralateral, normal lung. During expiration, the process was considered to reverse, gas passing from the contralateral lung into the lung underlying the paradoxing area (which appeared to expand). Fig. 1 illustrates the common theory of *pendelluft* as it is taught to medical students and presented in most surgical textbooks.

The concept of *pendelluft* forms an appealing theory to explain the clinical distress which occurs with paradoxical respiration. In fact, the *pendelluft* theory is so simple and neat that it has been accepted as fact without ever having been proved to be true.

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THEORETICAL EVALUATION

The argument which is basic to the *pendelluft* theory postulates that the affected lung is diminishing in volume at a time when the contralateral lung is expanding. Cursory examination of a patient with paradoxical respiration would tend to confirm this opinion, i.e., as the chest expands, the paradoxing area sinks inward. However, *more careful consideration suggests that in paradoxical respiration both lungs are actually expanding and contracting at the same time.*

Let us consider the actual area of paradoxical movement. With each inspiration, the chest wall sinks inward. Such an area will sink inward only if the pressure outside the chest becomes more positive, or if the pressure within

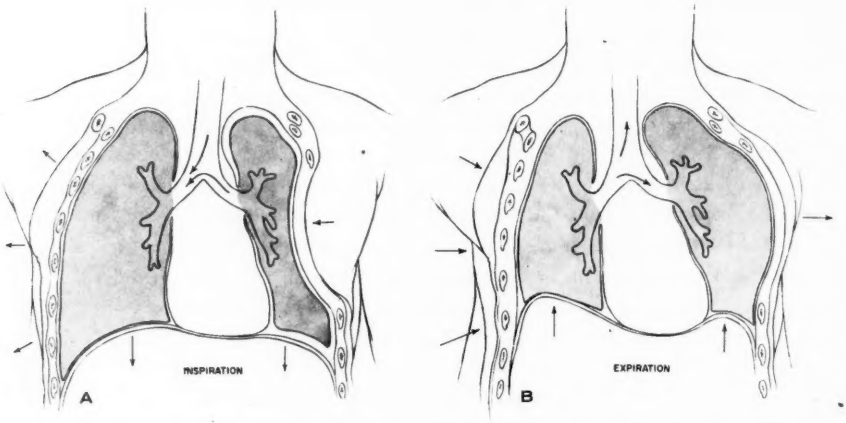

Fig. 1.—Schematic demonstration of paradoxical respiration and *pendelluft* as commonly depicted. An area of the left thoracic wall is decostalized. *A*, Inspiration: movement of alveolar gas from paradoxing lung to healthy lung. *B*, Expiration: movement of alveolar gas from healthy lung to paradoxing lung.

the pleural space becomes more negative. Since the chest is constantly exposed to unvarying atmospheric pressure, one must conclude that during inspiration pleural pressure underlying the area of paradoxical movement becomes more negative. During normal ventilation, the pressure within the lung itself remains essentially at atmospheric pressures.* It is quite apparent, therefore, that with inspiration the differential pressure across the lung increases (as the pleural pressure becomes more negative). Since the lung is an elastic body, increasing in volume with increasing differential pressures, it is clear that, despite the paradoxical movement of the chest wall, the affected lung actually increases in volume with inspiration. A schematic presentation of this discussion is given in Fig. 2.

Further evidence that *pendelluft* does not occur in the presence of paradoxical respiration can be derived from differential bronchospirometric studies

*For the purpose of this discussion, the small changes in intra-alveolar pressure due to the resistance of air flow are neglected.[2]

done in man by one of us (J. V. M.), in association with Sarnoff and Gaensler.[3] In a series of patients, ventilation of each lung was measured separately by means of differential bronchospirometry. The patients were then ventilated by electrophrenic respiration applied to one phrenic nerve. Despite the fact that respiration was carried out by one hemidiaphragm only, and despite the fact

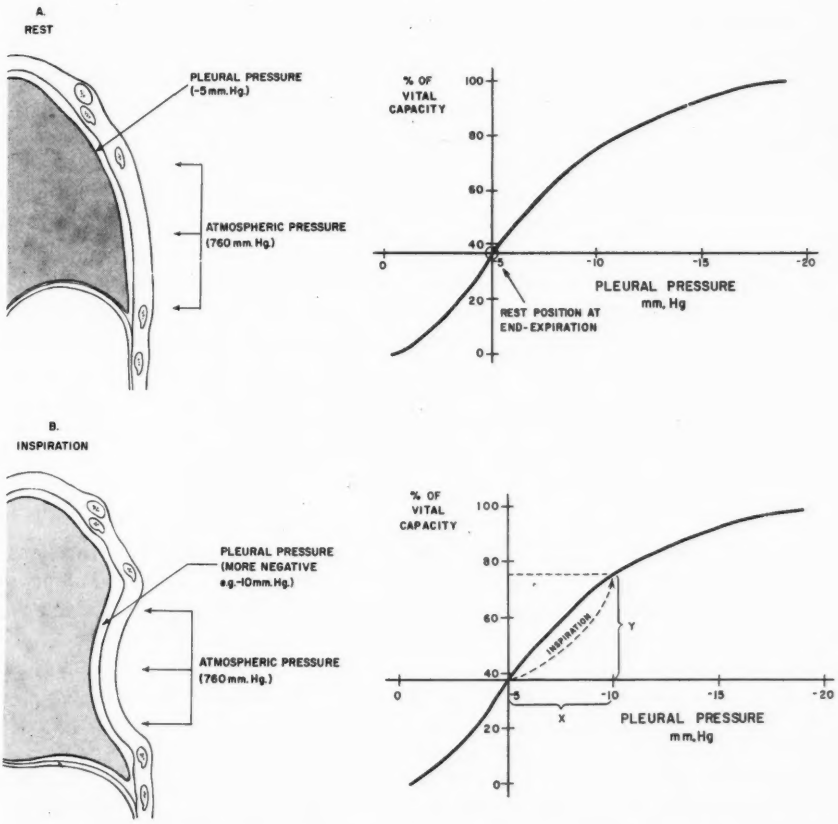

Fig. 2.—Schematic representation of paradoxical respiration and correlation with the pressure-volume curve of the lung. *A*, Lung in resting position. The decostalized portion of the chest wall is on the left. At the resting, end-expiratory position, plural pressure is −5 mm. Hg. *B*, During inspiration, the decostalized area sinks inward while the remainder of the chest is expanding. Since no change in atmospheric pressure occurs, the pleural pressure must be more negative to account for this paradoxical motion of the chest wall. The pressure-volume curve demonstrates that with inspiration and a more negative pleural pressure, the lung actually expands. If the lung on the paradoxing side expands during inspiration, there can be no *pendelluft*.

that there was marked paradoxical movement of the opposite hemidiaphragm, the lung on the paradoxing side continued to contribute its normal share of the total ventilation. The explanation of this observation lies in the fact that both lungs behave as if they were suspended in a single pleural chamber. Mediastinal

shift occurs to equalize the two pleural pressures, so that each lung continues to contribute its normal fraction of the total ventilation.

On theoretical grounds alone, it is possible to predict that, in the presence of a closed chest, *pendelluft* does not occur with paradoxical respiration.

EXPERIMENTAL EVALUATION

If *pendelluft* occurs, as illustrated in Fig. 1, the alveolar carbon dioxide from the paradoxing side should pass into the normal side during inspiration. During expiration, alveolar carbon dioxide would pass from the normal lung into the paradoxing lung. Therefore, it should be possible to detect this abnormal flow of carbon dioxide with the use of the currently available high-speed infrared gas analyzers. For this reason, a series of experiments was carried out before and after the performance of an extrapleural thoracoplasty in dogs.

Since the mediastinum of the dog is more tenuous and mobile than that of man, some objection may be raised as to the application of these studies to clinical situations. However, the bronchospirometric studies performed previously in 7 individuals[3] clearly demonstrated that the human mediastinum is completely mobile at resting rates of respiratory exchange, and that this structure produces no effective separation between the right and left pleural cavities in man under these conditions. The same situation obtains in dogs, as will be demonstrated subsequently. Therefore, the dog is, within these limitations, a satisfactory animal for the investigation of *pendelluft*.

METHODS

Five unselected, healthy mongrel dogs were anesthetized with intravenous sodium pentobarbital, 30 mg. per kilogram of body weight. With the animals lying supine on the operating table, an endotracheal tube with an inflatable cuff was passed. A pneumotachograph was used to measure the direction and rate of air flow through the endotracheal tube. A plastic catheter of 1.0 mm. internal diameter was threaded into both the right and left main-stem bronchi for gas sampling. By means of a constant speed pump, a continuous sample of gas was drawn from both main bronchi through a Liston-Becker Model 16* infrared gas analyzer (microsampling tube). Blunt cannulas were placed in both pleural spaces to measure intrapleural pressure. An electromechanical transducer was used to record motion of the chest wall. Appropriate transducers were employed to permit the simultaneous recording of the various parameters on a direct-writing, multichannel oscillograph. The experimental arrangement is illustrated in Fig. 3.

As a control observation, a recording of the following parameters was made during spontaneus respiration: infrared analysis of endobronchial carbon dioxide, right and left side; pneumotachogram; right and left pleural pressures; and motion of the chest wall. After this record had been obtained, a 3- or 4-rib

*Beckman Instrument Co., Palo Alto, Calif. The response time of the instrument to changes in carbon dioxide concentration at the end of the endobronchial catheter was 0.1 second.

extrapleural thoracoplasty was performed on the left side (ribs 3 to 6). All measurements were then repeated and the recordings examined for evidence of *pendulluft*.

RESULTS

The results of the studies of air flow, carbon dioxide, pleural pressure, and chest motion, before and after extrapleural thoracoplasty, were the same in all 5 animals. A representative sample of the tracing obtained before and after thoracoplasty is presented in Fig. 4. The pneumotachographic pattern is similar before and after thoracoplasty, although the respiratory rate is increased following the surgical procedure (channel 2). Channel 5 demonstrates the development of a severe degree of paradoxical motion of the chest wall following thoracoplasty. Pleural pressure recordings (channels 3 and 4) demonstrate that

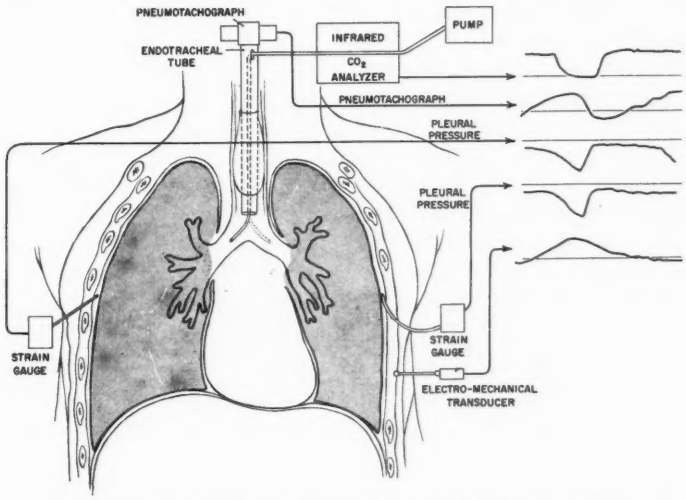

Fig. 3.—Illustration of recording system used to detect *pendelluft*.

the peak negative pressure on the side of the thoracoplasty is the same as that on the contralateral side. *This normal negative pleural pressure demonstrates that the lung on the side of the paradoxical respiration is actually expanding during inspiration.* This clearly eliminates the possibility of *pendelluft* and confirms the theoretical examination of the problem cited above.

The final proof of the absence of *pendelluft* in the presence of paradoxical respiration is found in the analysis of the endobronchial carbon dioxide concentration (channel 1). Correlation of the endobronchial carbon dioxide concentration and the pneumotachogram prior to thoracoplasty indicates that the concentration of the gas in the right main-stem bronchus reaches zero as soon as the inspiratory effort has cleared alveolar gas from the upper airway. It is important to note that after thoracoplasty the same pattern of bronchial carbon dioxide concentration obtains despite the presence of paradoxical respiration.

If *pendelluft* were to occur, the gas being inspired into the right lung would be continuously mixed with carbon dioxide passing over from the left main-stem bronchus. The carbon dioxide recording demonstrates that this is not the case. Examinations of the carbon dioxide concentration in the left stem bronchi during inspiration also failed to show evidence of *pendelluft*.

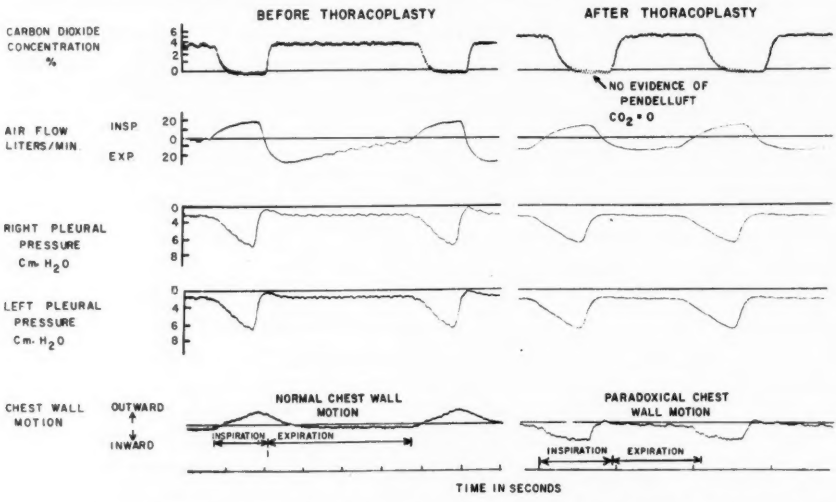

Fig. 4.—Multichannel recording of the various parameters investigated before and after extrapleural thoracoplasty on the left side. After thoracoplasty, respiratory distress is indicated by an increase in alveolar carbon dioxide concentration and an increase in respiratory rate (note change in time base). Despite the appearance of paradoxical respiration (channel 5) on the left side; pleural pressure in the left (channel 4) continues to make negative excursions as before. Therefore, expansion of the left lung during inspiration eliminates the possibility of *pendelluft*. Direct measurement of the carbon dioxide concentration in the right main bronchus (channel 1) shows no evidence of contamination of the inspired air with carbon dioxide from the contralateral lung.

DISCUSSION

The experimental data just cited give firm support to the theoretical analysis of the problem of *pendelluft*. Both in theory and under experimental conditions it is demonstrated that *pendelluft* does not occur in the presence of paradoxical respiration in the closed chest of the dog. According to the clinical studies quoted above, the results of these theoretical and experimental evaluations are pertinent to man as well.

There is one situation in which the surgeon occasionally may observe true *pendelluft*. This occurs in the presence of an open hemithorax with the patient breathing spontaneously. If the anesthesia bag is overdistended with gas, an unusual set of circumstances occurs. When the patient makes a spontaneous inspiratory effort, some gas will pass from the anesthesia system into the lung in the closed hemithorax. The pressure on the distended anesthesia bag will thereby diminish, and the pressure within the alveoli of both right and left lungs will decrease. Since atmospheric pressure (rather than pleural pressure) surrounds the lung in the open hemithorax, the lung will actually decrease in

volume while the contralateral lung increases in volume. Conversely, when the patient spontaneously makes an expiratory effort, the anesthesia bag becomes more distended and intra-alveolar pressure rises. Again, since the lung in the open hemithorax is exposed to a constant atmospheric pressure, the elevation of alveolar pressure in the exposed lung will cause this lung to expand while the contralateral lung diminishes in size. Thus, true *pendelluft* may occur with a spontaneously breathing patient in the presence of an open pneumothorax and an overdistended, closed breathing system.

The concept of *pendelluft* was invented to serve as a physiologic explanation for the respiratory distress which is observed following extensive thoracoplasty. However attractive this theory may be, the present study demonstrates that it is not adequate to explain the clinically observed distress seen in such patients. The physiologic deficit in these patients is actually related to the alteration in the pressure-volume curve of the chest (Fig. 2). As more of the chest wall is decostalized, the maximum degree of negative pressure which the patient can produce in a pleural space also diminishes. The patient under these circumstances lacks the mechanical ability to produce adequate pulmonary ventilation. Gaensler and Strieder,[4] in a study of patients before and after thoracoplasty, demonstrated this marked diminution in vital capacity and maximum breathing capacity. Furthermore, Björkman[5] demonstrated by differential bronchospirometry that the loss of function following thoracoplasty is attributable to a decrease in ventilation, not only on the side of operation but on the healthy side as well. From the studies cited, it is apparent that there is adequate explanation for the respiratory distress associated with paradoxical respiration without the invocation of the concept of *pendelluft*.

SUMMARY

Pendelluft, the pendulum-like movement of air from one lung to the other, is generally accepted as the cause of respiratory distress in the presence of paradoxical respiration. Despite the wide acceptance of this theory, no evidence has ever been presented in its support. The present study examines from both the theoretical and experimental points of view the subject of paradoxical motion of the chest wall and *pendelluft*.

An analysis of paradoxical chest wall motion in relation to the pressure-volume curve of the lung demonstrates that *pendelluft* does not occur in the presence of a closed chest. In an experimental study, simultaneous recordings of endobronchial carbon dioxide concentration, air flow, right and left pleural pressures, and chest wall motion were made before and after extrapleural thoracoplasty. The analysis of data from these experiments shows no evidence of *pendelluft*, despite the presence of paradoxical motion of the chest wall. Evidence is cited to support the applicability of these experimental studies to clinical situations.

On the basis of this study, it is recommended that the concept of *pendelluft* in the presence of a closed chest be abandoned.

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THE IMPORTANCE OF ANGIOGRAPHIC DIAGNOSIS IN INTRALOBAR PULMONARY SEQUESTRATION

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IT IS the purpose of this paper to record a case of intrapulmonary sequestration in which the correct diagnosis was established preoperatively by the method of retrograde aortography and to offer suggestions regarding surgical management.

The early clinical reports of Harris and Lewis[24] in 1940 and Pryce[35] in 1946 have emphasized the existence of a systemic arterial vessel from the aorta, or one of its tributaries, to the pulmonary lesion. Since this systemic arterial component may be of very large caliber, relatively short, and occasionally even multiple, it presents sometimes a real problem in management and a possible threat to the life of the patient during surgery. The candid reports of operative fatalities by Harris and Lewis,[24] Butler,[11] and Douglass[17] have served a very useful purpose in pointing out the technical hazards posed by the systemic arterial supply, and the importance of establishing, if possible, the diagnosis before surgery.

Presently available laminagraphic and especially angiographic techniques, which have been perfected in the study of other pathologic disease, can also provide precise delineation of the arterial pattern in a pulmonary sequestration. The aid which this type of advance knowledge may lend to the planning and safe execution of excisional procedures is obvious.

CASE REPORT

J. M. (unit No. 51-27-37), a 27-year-old Negro man, was hospitalized as a result of findings in a pre-employment x-ray examination of the chest. He had always considered his health to be good except that 2 years previously he had had "virus pneumonia" associated with cough, chills, and fever during an epidemic of influenza. His symptoms at the time of this admission were cough and the production of thick green sputum amounting to about a cupful per day over a period of at least several months. He denied dyspnea, hemoptysis, and chest pain.

Examination revealed a slender, Negro man, weighing 120 pounds, with a blood pressure of 110/60 mm. Hg, pulse 100, and temperature 97.8° F. Pertinent findings included a shift of the heart and mediastinun to the right side. On auscultation there was

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a succession splash in the left lower chest. Breath sounds over this region were diminished and loud rhonci could be heard at times. An indirect left inguinal hernia was present, and there was early clubbing of the fingers.

Laboratory data included a hemoglobin of 13 Gm., hematocrit of 44 per cent, white blood count of 4,400. A test for sickling of erythrocytes was negative. Urinalysis revealed occasional one plus albuminuria. Blood chemistries showed a urea nitrogen of 10 mg. per cent, fasting blood sugar of 93 mg. per cent, total protein 8.5 Gm. per cent, albumin 2.6 Gm. per cent, and globulin 5.9 Gm. per cent. Later the total protein was 9.16 Gm. per cent with albumin 2.67 Gm. per cent and globulin 6.49 Gm. per cent. A test for lupus globulin was negative and Bence-Jones proteinuria was not present. The serum calcium was 10.6 mg. per cent and serum phosphorus was 4.4 mg. per cent. Serum bilirubin

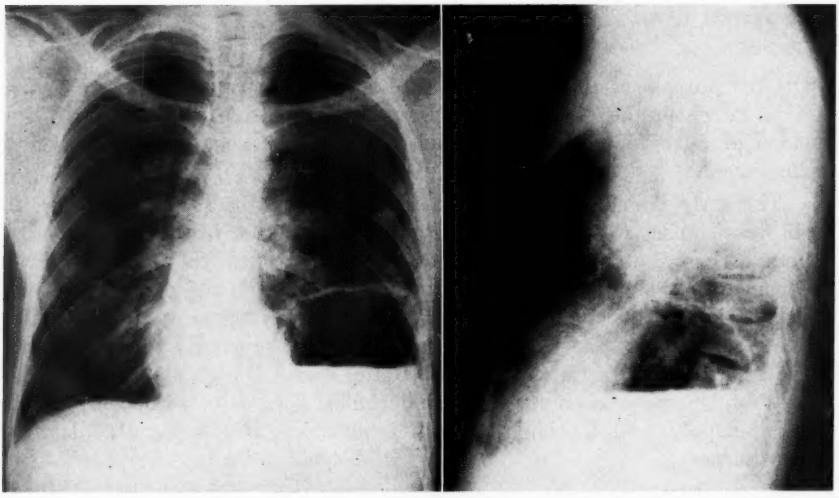

Fig. 1. Fig. 2.

Figs. 1 and 2.—Posteroanterior and lateral views of chest show a large cystic space in the left lower lobe with an air fluid level within it and shift of the mediastinum to the right.

was 0.56 mg per cent total, with 0.17 mg. per cent in one minute; Bromsulphalein retention 5.3 per cent in 45 minutes, cephalin flocculation negative, thymol turbidity 1.8, thymol flocculation 1+, alkaline phosphatase 5.0 Bodansky units. Sputum on culture yielded 3+ growth of pneumococci. An electrocardiogram was interpreted as showing increased voltage in the right precordial leads.

Chest x-ray studies (Figs. 1 and 2) showed a large, relatively thin-walled cystic space in the left lower lung which contained an air-fluid level. There was compression of surrounding lung and shift of the mediastinum toward the right. Laminagraphy showed septation of the cyst. Iodized oil bronchography revealed displacement of the segmental bronchi of the left lower lobe; several droplets of contrast material penetrated into the cystic space, although a communication with the bronchial tree was otherwise not visualized. Retrograde aortography through a catheter, passed from the left femoral artery into the thoracic aorta, showed a large anomalous artery arising from the thoracic aorta and supplying the region of the cyst (Figs. 3 and 4).

At operation on July 9, 1959, there were dense adhesions about the left lower lobe, especially over its diaphragmatic and mediastinal surfaces. The descending aorta was mobilized and surrounded with an umbilical tape proximally so that it could be temporarily occluded should the need arise. A large, relatively normal-appearing artery arose from

the descending aorta just above the diaphragm and passed within the leaves of the pulmonary ligament to the medial border of the left lower lobe. A vein of smaller size accompanied the artery and drained into the hemiazygos vein. A left lower lobectomy was performed from which recovery was essentially uneventful.

Pathologic Study.—On inspection of the resected lobe, the anomalous artery and vein could be seen entering the posteroinferior mediastinal surface. In the central portion of the lobe was a large trabeculated cystic space containing purulent material. Microscopic examination of the proximal segment of the anomalous artery showed a normal systemic arterial structure without evidence of atherosclerosis.

After injection of the arteries, veins, and bronchi with Vinylite and acid digestion of the tissue, some communications of the anomalous systemic artery with the pulmonary arterial system were demonstrated, as well as between the systemic vein and pulmonary veins about the cyst.

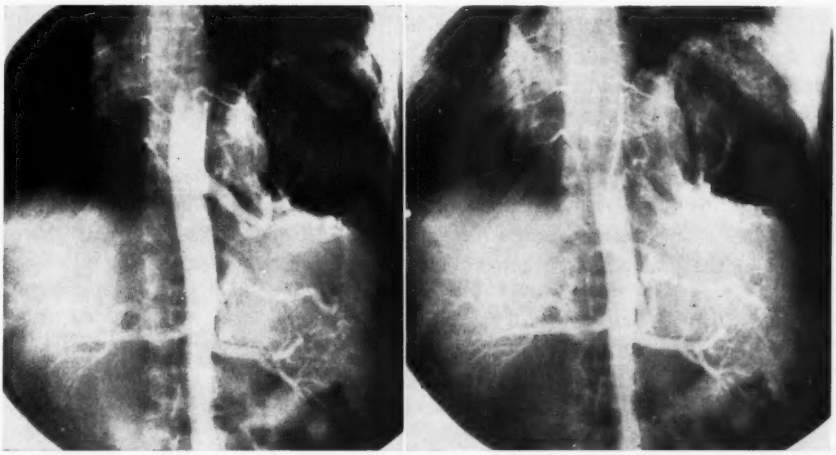

Fig. 3. Fig. 4.

Figs. 3 and 4.—Early and later films from the retrograde aortogram showing the anomalous systemic artery arising from the distal thoracic aorta and ramifying about the sequestration.

DISCUSSION

The embryologic, pathologic, and clinical pictures of intralobar pulmonary sequestration have been well presented by several authors and need not be repeated in detail. The developmental error occurs at an early stage in embryonic life. As the lung bud develops ventrally from the primitive foregut, one of its subsequent branches apparently becomes detached and acquires systemic arterial blood supply, probably by the persistence of some part of the splanchnic plexus. Whether the abnormal vascular supply is indeed the cause rather than the result of the sequestration, as suggested by Pryce,[35, 36] is not known. As further development proceeds, the systemic vascular supply component usually moves caudally, accompanying the migrating pulmonary tissue. Since the diaphragm completes its development somewhat later, the anomalous vessel may arise either above or below it.

Bronchial secretion within the sequestered elements leads to the development of fluid-filled cystic spaces. The lesion may then be present for an indeterminate period and, unless discovered by x-ray examination, does not come to clinical attention because symptoms are absent. However, if infection takes place, symptoms can arise; usually a communication with the more normal bronchial tree becomes established, producing purulent sputum and an air-fluid level within the cystic spaces.

Unless a proper diagnosis is made, futile attempts at external drainage and delay in the indicated resectional procedure may take place. When consideration of surgical treatment arises, the anomaly of arterial blood supply assumes major importance, and relatively less attention perhaps has been given this aspect of the condition in the literature.

The present case, added to others in the English bibliography, gives 114 surgical cases in which the description of the vascular pattern to the sequestration is adequate for analysis. This summary does not include instances of anomalous pulmonary arteries unassociated with sequestration, or cases of extralobar sequestration.

As shown in Table I, the left lower lobe is the site of sequestration in about two thirds of the cases, with about one third in the right lower lobe. Only rarely does sequestration occur in the middle and upper lobes. Roughly three fourths of the patients were under 30 years of age when operated upon.

TABLE I. SUMMARY OF SOME REPORTED OBSERVATIONS IN INTRALOBAR PULMONARY SEQUESTRATION

Location

Lobe	Left lower	Right lower	Right upper	Left upper	Unknown	Total
No. of cases	67	36	2	1	8	114

Age at Operation

Age range (years)	0-10	10-20	20-30	30-40	40-50	50-60	60+	Unknown	Total
No. of cases	28	23	27	13	6	6	3	8	114

Origin of Anomalous Systemic Artery

Origin	Thoracic aorta	Below diaphragm	Intercostal	Aortic arch	Multiple	Not stated
No. of cases	74	25	5	1	17	9

Size of Anomalous Systemic Artery

Diameter	<0.4 cm.	0.4-1.0 cm.	More than 1 cm.	Not stated	Multiple vessels
No. of cases	5	51	5	36	17

Microscopic Characteristics of the Arterial Wall in 23 Cases

	"Elastic" type	"Combined" type	"Systemic" type
No. of cases	12	6	5

Atherosclerosis

	None	Mild	Moderate	Severe
No. of cases	9	2	9	6

The site of origin of the anomalous artery is usually the descending aorta immediately above the diaphragm. Origin below the diaphragm, usually from the abdominal aorta, is less common, occurring, however, in almost one fourth

of the cases. Vessels arising from the intercostal arteries are more rare. Multiple tributary vessels, sometimes as many as five or six, have been recorded in at least seventeen instances.

The course of anomalous vessels is quite variable, but most commonly they enter the lung through the pulmonary ligament. They may, however, be encountered on the diaphragmatic and lower posterior surfaces of the lower lobes in addition to the mediastinal surface. An exact knowledge of the origin, caliber, and course of the anomalous systemic artery in these patients is of obvious importance. In almost all cases, the pleural space is obliterated by adhesions in the region of the sequestration, making identification of the tributary vessel difficult. In some cases, there are projections of pulmonary tissue along the anomalous artery into the mediastinum, tending to obscure the vessel and making its dissection hazardous.

The caliber of the aberrant artery may be surprisingly large, especially in the young children reported. The aortogram in the present case (Figs. 2 and 3) demonstrates that the vessel is considerably larger than the renal arteries. Vessels up to 2 cm. in diameter have been reported, but the vast majority are between 4 and 10 mm. in diameter. If the arteries are less than 3 mm. in diameter they are likely to be multiple.

Veins accompanying the anomalous artery and which join in the systemic return to the right heart have been described in only six instances, including the presently reported case. These veins have generally been considerably smaller than the artery, suggesting that only a portion of the venous blood returns from the sequestration to the right heart and that some at least enters the pulmonary venous return to the left side of the heart. In the more usual situation, i.e., without systemic venous branches, almost all the venous return is by way of pulmonary veins, thus producing a systemic arterial-left atrial shunt. As far as we are aware, however, no physiologic data have been obtained to prove the existence of such a shunt and to show its hemodynamic effects.

In two reported instances, fibers from the splanchnic nerves were demonstrated accompanying the anomalous artery into the lung substance.

The available descriptions of the gross and microscopic appearance of the anomalous arteries indicate that these vessels tend to be of the "elastic" type. In some cases, the artery is predominantly "muscular" near its origin but becomes "elastic" peripherally. Some vessel walls have been described as closely resembling the structure of a pulmonary artery, some as being "hybrid" in nature and others similar to systemic visceral arteries or miniature aortae. These vessels have shown in many instances atheromatous changes, often in relatively young individuals. In a few cases, atheromatous changes occurred in those adjacent pulmonary arterial branches which were thought to anastomose with the anomalous systemic artery. Only a single instance of thrombosis in one of these diseased vessels could be found.[4]

We are persuaded that if the details of the anomalous vessel or vessels are known, a safer surgical attack can be planned. If the anomalous artery originates from the aorta itself, dissection and mobilization of the aorta sufficient

for the possible emergency application of a suitable clamp proximal to the origin of this vessel are indicated. If the vessel originates from an intercostal or other artery appropriate proximal control should also be gained *prior* to the dissection of the anomalous artery and the lobe itself.

Great care should be taken in securing the anomalous artery by clamps or ligatures lest a markedly atherosclerotic vessel be torn or fractured. Palpation of the pulmonary ligament for pulsations prior to its division should be routine in every lobectomy, since anomalous vessels occasionally occur without an evident sequestration. When at surgery the pulmonary artery or one of its major branches seems unduly small, the possibility of an anomalous vessel from the systemic circulation should be kept in mind, and appropriate exploration made to exclude it.

SUMMARY

A case of intralobar pulmonary sequestration is described in which preoperative retrograde aortography demonstrated the anomalous arterial supply.

Information is summarized from the literature concerning the origin, course, caliber, and structural characteristics of anomalous arteries related to intralobar sequestrations.

The danger of an operative injury to the anomalous vessels is again pointed out. The desirability of a preoperative demonstration of the origin, course, and caliber of these vessels by radiographic techniques is stressed.

Surgical safeguards, including dissection and mobilization of the descending aorta, when appropriate, before an approach to the anomalous vessel itself, are recommended.

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LOCALIZED EMPHYSEMA IN ASSOCIATION WITH BRONCHIAL CYSTS OR MUCOCELES

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LOCALIZED pulmonary emphysema in association with bronchial cysts or mucoceles has not been previously reported in the literature. The localized emphysema associated with bronchi which are partially obstructed by cicatrization, neoplasia, or foreign bodies is well known. In these cases the demonstrable obstructing mechanism causes distal emphysema. Such emphysema is the result of the valve-like action caused by the obstructing lesion, which permits inflow of air during inspiration but obstructs the flow during expiration.

Large emphysematous areas, or bullae, within lung tissue should not be termed cysts, since they contain air. The term ***bronchial cyst*** identifies a fluid-containing sac, relatively spherical in shape, which is lined by respiratory tract epithelium. The term *mucocele* refers to a branched and dilated bronchial structure containing fluid and lined by respiratory tract epithelium. Bronchial cysts and mucoceles are similar abnormalities of the bronchial tree. Their outline differs, however, since they involve either a single segment of a bronchus or a branched portion of a bronchus and its subdivisions. These cysts and mucoceles presumably represent developmental abnormalities of the bronchial tree. They are isolated from the pulmonary parenchyma distally and do not serve in a ventilatory air-conducting capacity for the surrounding lung tissue. The cysts or mucoceles, in general, are completely isolated from the major tracheobronchial tree proximally, although some cysts and mucoceles do have minute proximal communications with the tracheobronchial tree. As a result of this communication they may show air-fluid levels within them. The fluid within the cysts or mucoceles is excreted by the mucous glands within the wall of the cystic structure.

It is believed that budding of the tracheobronchial tree into the mesenchyma of the lung begins after the fourth week of fetal life and may continue until the fourteenth year after birth.[1, 2] A disturbance at any point in the development of the 24 orders of bronchial subdivisions that occurs from the fourth week of intrauterine life to adolescence can result in a bronchial abnormality. Bronchial cysts or mucoceles, therefore, may be classified as either congenital anomalies or postnatal developmental malformations.

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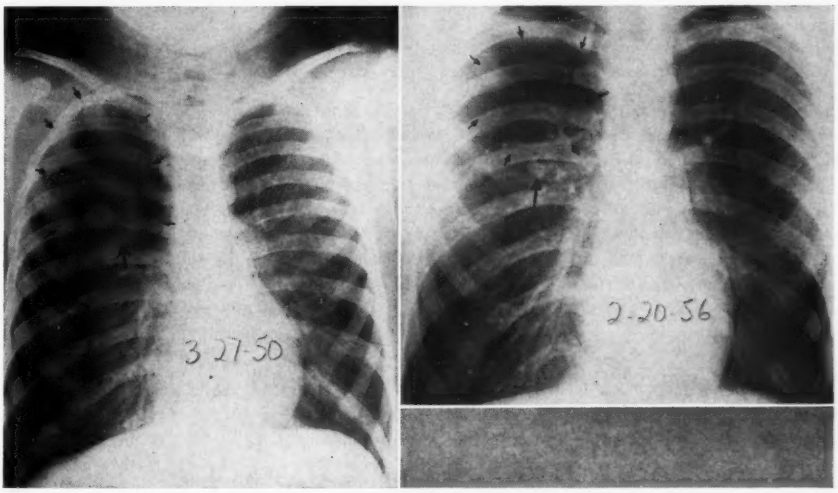
The syndrome of localized pulmonary emphysema in association with bronchial cysts or mucoceles is illustrated by the following case reports. In 2 of the patients, excisional procedures were carried out and the specimens demonstrated the pathologic changes involved. In the third patient the diagnosis was made on radiologic evidence alone.

CASE REPORTS

CASE 1.—A 15-year-old white boy developed pneumonia in the posterior segment of the right upper lobe of the lung in January, 1956. All the roentgenograms made during and following the acute episode demonstrated emphysema localized to the apical and anterior segments of the right upper lobe (Fig. 1, *B*). A chest film taken 6 years previously demonstrated emphysema in the same area (Fig. 1, *A*). A 1.5 cm. rounded opacity in the right parahilar area was present in all films.



A. *B.*

Fig. 1.—Case 1. *A*, In a roentgenogram taken March 27, 1950, emphysema is demonstrated in the right upper lobe. The large arrow points to a mass in the second right anterior interspace. *B*, Six years later, emphysema is again demonstrated. Note the well-defined mass now evident in the second right anterior interspace *(large arrow)*.

The patient's history indicated reduced respiratory reserve. Surgical exploration was elected to remove the apparently expanding area of emphysema and to investigate the mass. A right upper lobectomy was carried out on June 18, 1956.

Pathologic Examination.—A 3.2 cm. cystic mass in the right parahilar area was filled with mucoid secretions. The lung showed macroscopic and microscopic emphysema. The bronchial cystic structure was lined with respiratory epithelium. The walls of the cyst contained cartilage, and mucous and serous glands. There was practically no pigment in the interstitial tissue or in the macrophages in the alveoli.

CASE 2.—A 41-year-old Negro man had a routine chest film which showed emphysema of the right upper lobe of the lung and an opaque parahilar mass (Fig. 2, *A*). The patient was asymptomatic. He was admitted to the hospital on Oct. 22, 1958, for investigation and treatment.

The results of bronchoscopy were negative. The orifices of the three segmental bronchi of the right upper lobe were visualized with a telescope and were normal. Bronchograms showed an obstruction of the bronchus of the anterior segment at a point just beyond its orifice (Fig. 2, *B*). A large mass was seen adjacent to the obstructed bronchus. Tomograms showed that the large parahilar mass was accompanied by several smaller opaque masses extending out anteroinferiorly. In all films the masses lay within the emphysematous lung tissue. The segmental bronchi that filled with contrast material branched into normal-appearing lung tissue.

On Nov. 12, 1958, thoracotomy showed that the anterior segment of the right upper lobe was markedly emphysematous and was compressing the apical and posterior segments. The right middle lobe and the right lower lobe showed no emphysematous changes. The parahilar mass, described previously, contained clear viscid fluid. There were several smaller fluid-filled cystic masses extending anteroinferiorly. The lumen of the large cystic mass was not in continuity with the tracheobronchial tree, proximally or distally. The right upper lobe was excised. Microscopic sections showed cystic spaces lined by either columnar or pseudostratified columnar epithelium which rested upon laminated layers of fibrous tissue (Fig. 2, *C*). There was no cartilage in the fibrous tissue. The surrounding pulmonary parenchyma was extremely emphysematous and contained a very few, widely scattered, pigment-containing macrophages. In some regions the pulmonary parenchyma was atelectatic; here the bronchi were normal in caliber and architecture.

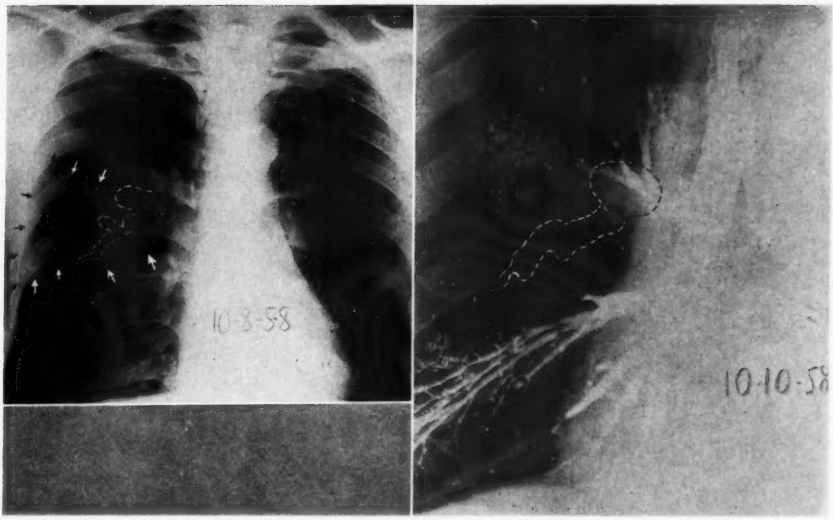
CASE 3.—A 25-year-old white man had a routine roentgenogram which revealed a small right parahilar mass and, peripheral to this, an area of pulmonary emphysema (Fig. 3, *B*). The patient was asymptomatic. The similarity in radiologic appearance to the previous 2 cases suggested the diagnosis of pulmonary cyst or mucocele in association with pulmonary emphysema. Review of an x-ray film of May 27, 1954, showed a lobulated mass in the right parahilar area and, surrounding this mass, emphysematous tissue in the right upper lobe (Fig. 3, *A*). The amount and degree of emphysema at that time was much less marked.

Roentgenograms showed a 1.5 cm. parahilar mass with an air-fluid level within it on the routine upright film. Films in the lateral decubitus projection showed a shift of the air-fluid level. Posteroanterior and lateral tomography (Fig. 3, *D* and *E*) showed a cystic structure, irregular in outline, which measured up to 4 cm. in its greatest diameter. This structure appeared to lie within the superior segment of the right lower lobe. Expiration films (Fig. 3, *C*) demonstrated the obstructive air-trapping phenomena of pulmonary emphysema. The adjacent right lower lobe and right middle lobe were compressed and the mediastinum was shifted to the left. This was best seen on the bronchograms taken during expiration. The bronchograms showed apparent filling of all segmental bronchi, but there was no flow of the contrast medium to the emphysematous parenchymal area.

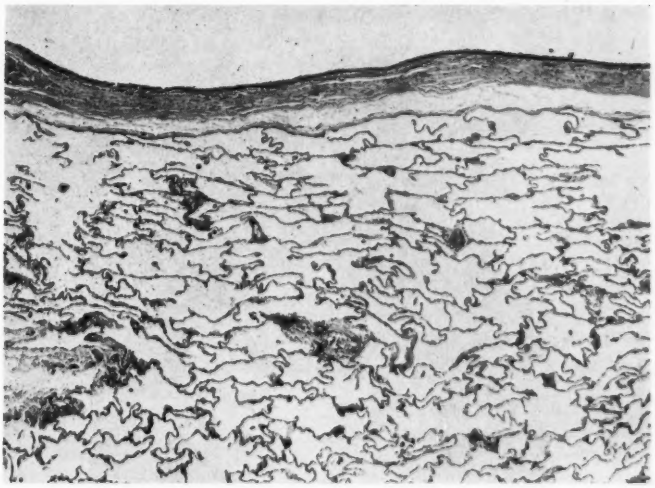
The filling of all segmental bronchi with contrast material indicated that the bronchial cystic architectural alteration occurred in a bronchial subdivision distal to a segmental bronchus. The demonstration of an apparently normal bronchogram, showing all segmental bronchi, falls short of being a true indication of a completely normal bronchial tree, since alterations of architecture of the bronchi beyond the tertiary bronchi are difficult to discern.

DISCUSSION

In 1931, Van Allen and Lindskog[3] described collateral respiration in the lung and experimentally demonstrated that adjacent lung lobules (segments and subsegments) were connected by collateral interalveolar pathways which permitted gases, particulate matter, and thin fluids to cross between the lobules. These authors further demonstrated some interesting facts relative to segmental bronchial occlusion and collateral filling, and later segmental atelectasis.



A. *B.*



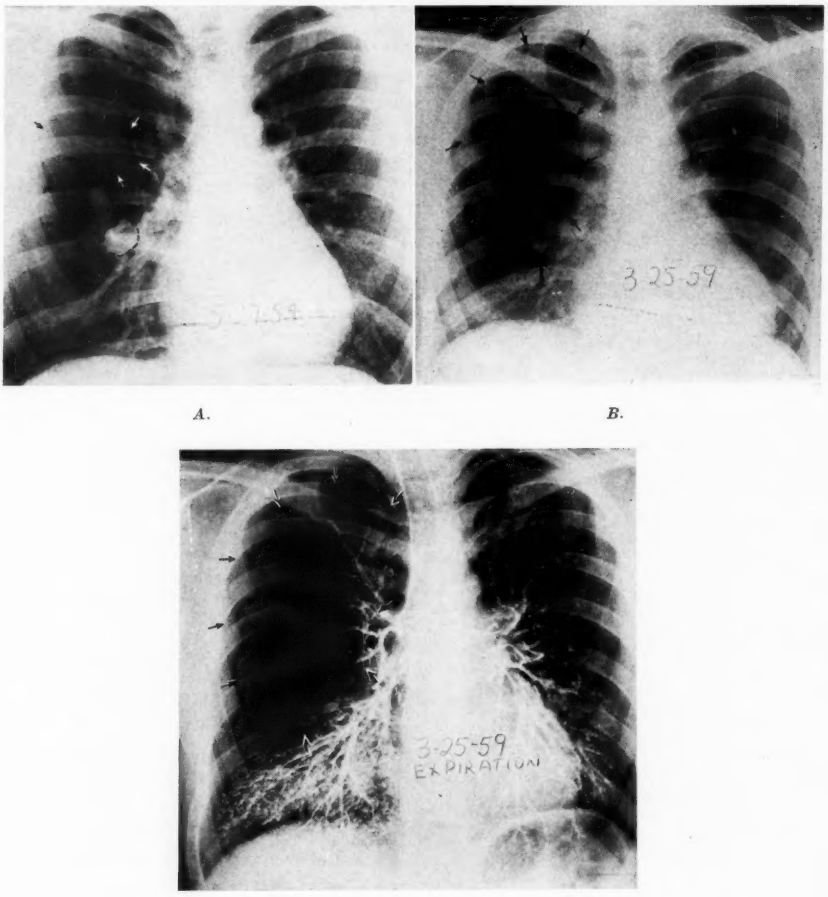
C.

Fig. 2.—Case 2. *A*, The emphysema is localized to the right mid-lung area. An irregularly outlined mass is demonstrated in the right third anterior interspace, extending anteroinferiorly into the emphysematous lung tissue.

B, The anterior segmental bronchus of the upper lobe does not fill with radiopaque material. The mucocele extends mediolaterally from the hilar area toward the inferior portion of the emphysematous tissue.

C, The photomicrograph demonstrates the epithelial lining of the mucocele. The mucocele is surrounded by emphysematous pulmonary parenchyma.

Sudden occlusion of a segmental bronchus does not ordinarily cause immediate atelectasis. The air content of the occluded segment is temporarily maintained by air drift through the collateral pathways from adjacent nonobstructed segments. The bronchi and bronchioli distal to the point of obstruction continue to secrete mucus normally from their lining of epithelium. These secretions then flow in a retrograde direction to enter the alveoli and thus impede continued air drift. Simultaneously the air within the alveoli is absorbed. Thus,



A. *B.*

C.

Fig. 3.—Case 3. *A*, A roentgenogram taken April 27, 1954, shows emphysematous tissue in the right mid-lung. Extending into this area is a branched mucocele.

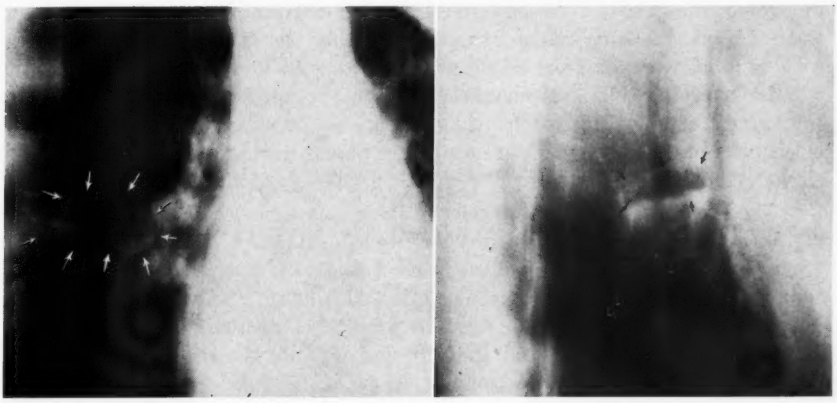
B, The film taken March 25, 1959, 5 years later, shows that the emphysematous area has enlarged. The mass in the third right anterior interspace shows an air-fluid meniscus *(large arrow)*.

C, An expiration film clearly demonstrates the air-trapping phenomena of the emphysematous tissue. There is mediastinal shift to the left as well as compression of the right middle lobe and right lower lobe segmental bronchi.

when its bronchus is obstructed, a form of consolidation-atelectasis of the involved segment occurs as a result of the absorption of air from, and the secretory filling of mucus into, the alveoli.

In contradistinction to this retrograde filling of the alveoli by bronchial mucus, Churchill[4] showed that with inflammatory or fibrous obliteration of the finer bronchial and bronchiolar radicals, the mucoid bronchial secretions cannot back up into the alveoli to fill them. Under these circumstances, air drift into the alveoli can be maintained indefinitely through the collateral pathways.

Huppler and associates[5] demonstrated in the experimental animal that when portions of a bronchus were occluded at two sites proximally and distally, little mucus was present in the parenchyma distal to the obstruction. They



D *E*

Fig. 3.—Cont'd. *D*, The posteroanterior tomogram clearly shows the mucocele and its air-fluid contents. *E*, The lateral tomogram again shows the mucocele, which is now demonstrated to be in the superior portion of the right lower lobe.

also showed that when the lobar bronchi were divided and closed at a single level, mucus was later found within the distal bronchi and within the alveoli that were peripheral to the occluded bronchus.

In the 3 cases presented here, localized pulmonary emphysema was found in association with bronchial cysts. In each case the emphysema was localized to a section of a lobe, and did not involve the entire lobe or the remainder of the lung. In these patients the bronchial cysts were isolated completely from the peripheral parenchyma and totally or partially from the proximal tracheobronchial tree. The obstruction distally of the cysts from the peripheral parenchyma isolated this parenchyma from its normal ventilatory pathway and also precluded the filling of the parenchyma with bronchial secretions. Thus, the development of lung drowning (filling with bronchial secretions) that generally follows pure proximal bronchial obstruction was prevented. Because of freedom from secretory drowning of the lung, collateral ventilation through the alveolar pores of Cohn maintained the expansion of the lung parenchyma indefinitely.

As a result of more efficient inspiratory filling than expiratory outflow of air through the collateral channels, obstructive emphysema occurred in this area of lung parenchyma.

Surgeons have noted the ease of filling of a segment of parenchyma even when the bronchus to that segment has been temporarily clamped. Yet collapse occurs less readily in that segment than in the adjacent nonobstructed segments of parenchyma. Similarly, patients who have segments of parenchyma isolated from their normal ventilatory pathways by cystic changes of the bronchi never have a normal expiratory collapse of the parenchyma. McLean[6] offers the hypothesis that the positive pressure of expiratory contraction of the lung causes collapse of the pores of Cohn, through which collateral ventilation occurs. During inspiration the expanding thorax and lung parenchyma maintain the patency of the pores. In essence, there is a ball-valve mechanism at the pores of Cohn. Thus, the lung parenchyma surrounding the bronchial cysts or mucoceles is filled with air from adjacent segments with normal bronchial conduits by means of the collateral channels through the pores of Cohn. Obstructive emphysema of these indirectly filled segments occurs as a result of the ball-valve mechanism at the pores of Cohn, which are patent during inspiration but which close during expiration.

The bronchial cysts or mucoceles in these 3 cases may well be developmental malformations, although this cannot be proved. Ramsay,[7] in a case report of a 15-year-old girl, described a multibranched mucocele with direct bronchial continuity between the superior segment of the left lower lobe and a posterior subsegment of the left upper lobe. The pathologic anatomy suggests that the condition was a developmental anomaly and was not the result of an acquired inflammatory bronchial obstruction.

This complex of bronchial cystic change in association with pulmonary emphysema presents a typical and readily identifiable radiologic picture. Localized pulmonary emphysema in association with a parahilar mass which may or may not show an air-fluid level should suggest the diagnosis. After the tentative diagnosis is made by roentgenography the problem of therapy may then be considered.

All 3 patients described here were relatively asymptomatic; only the first (the 15-year-old boy) had some clinical evidence suggestive of a decrease in respiratory reserve. This patient also had evidence of progressive expansion in the area of emphysema with resultant compression of the adjacent right middle lobe and right lower lobe.

Apparently excisional therapy need be carried out only (1) if the degree of encroachment of the area of emphysema upon adjacent normal lung tissue becomes clinically significant, or (2) if infection of the cystic area occurs and cannot be readily controlled and overcome by nonsurgical means, or (3) if infection is recurrent in the cyst (or mucocele).

There should be no difficulty in differentiating this complex from that of the coin lesion or mass of undetermined etiology in a patient with generalized emphysema. When a question exists whether a neoplasm is causing peripheral

emphysema, exploratory thoracotomy is indicated. However, when the complex has been known to exist for a number of years (as in Cases 1 and 2) and the patients are asymptomatic, exploration seems unnecessary.

SUMMARY

The association of localized emphysema with a bronchial cyst or mucocele presents a radiologically identifiable syndrome. The normal bronchial ventilatory pathways of the emphysematous portion of parenchyma have been altered by cystic changes. Aeration occurs from adjacent normal parenchymal segments through the collateral interalveolar channels of the pores of Cohn. The emphysema results from the obstructive ball-valve mechanism of the air conducting conduits, the pores of Cohn. Three case reports which demonstrate this syndrome are presented.

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INTRALOBAR ENTERIC SEQUESTRATION OF THE LUNG CONTAINING ABERRANT PANCREAS

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THE ASSOCIATION of a congenital cyst of the lower lobe of either lung with an anomalous artery arising from the aorta has been generally accepted to define the condition called "intralobar pulmonary sequestration." Extrapulmonary sequestrations with anomalous arterial supply and so-called "accessory lung or lobe" appear to have similar embryologic origin. However, their clinical and radiologic manifestations are different enough to classify them as separate entities.

The purpose of this report is to detail an unusual variant of an intralobar sequestration of the left lower lobe. The anomalous cystic lobe contained aberrant pancreatic tissue and was connected to the esophagogastric junction by an esophageal duplication, having all the histologic characteristics of esophagus, except for an absent lumen. A similar type of sequestration has been reported by Baar and D'Abreu.[1]

CASE REPORT

The patient was a Negro female infant, 14 months of age, with a history of severe chronic cough and intermittent upper respiratory infections since the age of 4 weeks. She was a frail infant, far below the normal percentile for her age. When 6 months old, she was hospitalized for 3 months and underwent medical treatment for what was believed to be a lung abscess of the left lower lobe. A radiolucent 4 cm. cavity was noted at that time within the left lower lobe. The child showed no improvement on intensive antibiotic therapy and surgery was deferred because of her poor condition.

When first seen at 14 months of age, the same thick-walled 4 cm. air-filled cavity within the posterior basal segment of the left lower lobe was demonstrated (Fig. 1). Moist râles were heard over both lung fields. Despite antibiotic therapy, the child ran a temperature elevation up to 101° F. Bronchoscopy disclosed intense reddening and granularity of the left main-stem bronchus, and bronchograms showed displacement of the left lower lobe basal bronchi by the cyst. No direct communication with the cavity was demonstrable. It was our opinion that the child had either a congenital cyst or a chronic lung abscess, and surgery was advised.

At operation, the left lower lobe was found to be airless and contracted, with overdistention of the normal left upper lobe. A large fluctuant cystic mass could be felt within the posterior basal segment of the lower lobe. Coming up through the esophageal hiatus and entering the diaphragmatic surface of the lower lobe, parallel to the inferior pulmonary ligament, was a 2 cm. muscular tubular structure having all the gross characteristics of esophagus.

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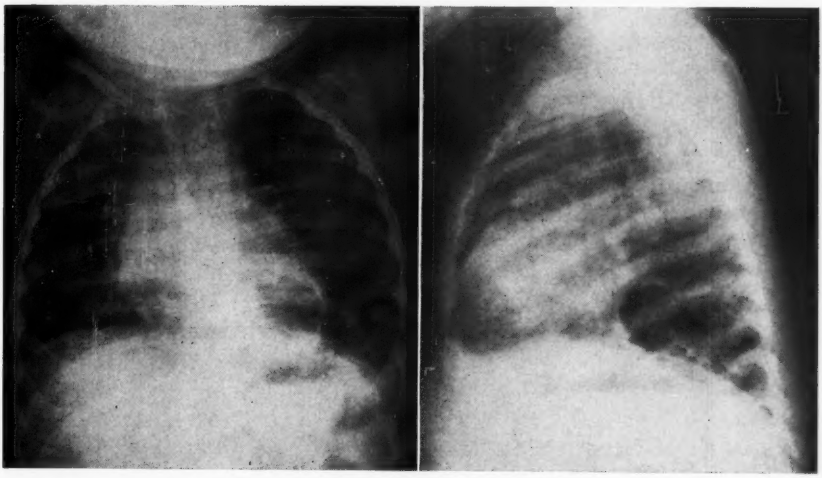

Fig. 1.—Posteroanterior and left lateral films of chest taken preoperatively which show the large cystic sequestration within the left lower lobe.

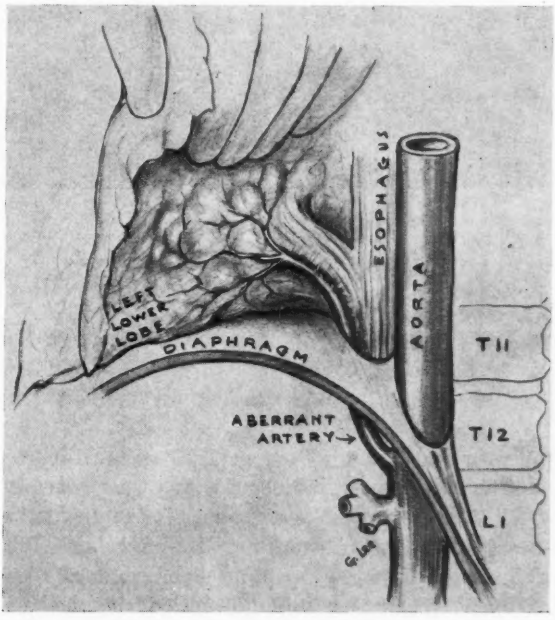

Fig. 2.—Illustration of sequestration, anomalous artery, and esophageal duplication as seen at operation.

Attached to this structure was a large 4 mm. artery, which, on dissection, arose from the abdominal aorta just above the celiac axis (Fig. 2). This artery branched as it entered the cystic area within the left lower lobe. The esophagus-like tubular structure was seen to arise from the esophagogastric junction as a separate entity.

The aberrant vessel was ligated and divided, the esophageal extension to the lower lobe was similarly divided, and the left lower lobe resected in the usual manner. There were no venous anomalies. The proximal end of the esophageal duplication was closed with interrupted fine silk, but was not demonstrated to have a patent lumen. The pulmonic end of the esophageal tube did develop a lumen and entered directly into the forementioned cyst. This esophageal duplication had all the gross and microscopic characteristics of esophagus with both squamous and gastric mucosal lining. There were several pink masses of tissue in the periphery of the cyst which startlingly were revealed to show secreting pancreatic tissue on histologic examination (Fig. 3).

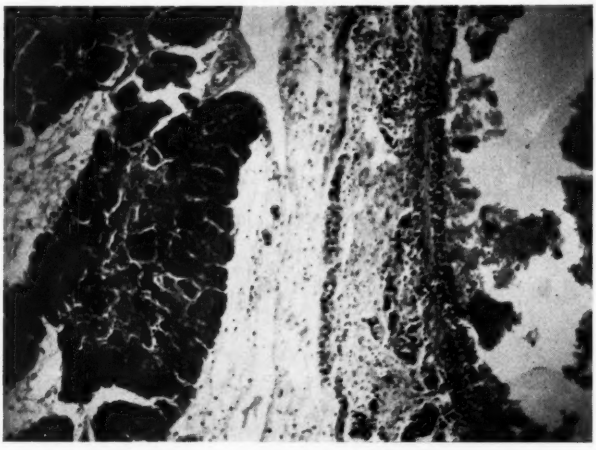

Fig. 3.—Photomicrograph (low power) which shows islands of aberrant pancreas within the wall of the congenital cyst. The characteristic columnar epithelial lining of the cyst can also be seen.

The child made an uneventful recovery following removal of the diseased left lower lobe, with dramatic disappearance of all symptoms and with rapid weight gain. At this writing, the child is 2 years postoperative, with no recurrence of pulmonary symptoms and with a normal chest x-ray.

DISCUSSION

Interest in this anomaly dates back to 1777 when Hubert described an isolated systemic arterial anomaly to the lung without associated bronchopulmonary pathology. Within the past 20 years, congenital sequestration has become of increasing interest to the thoracic surgeon. Almost all cases observed at the autopsy table or at surgery have shown various manifestations of single or multilocular congenital cystic structures within the affected lobe. There are either one or two aortic systemic branches which nourish these sequestered segments.

Scheidegger,[7] in 1936, reported a complete left accessory lung not connected to the lower lobe. This structure was found in the posterior gutter with no

bronchial communication to the lung, but connected to the cardia of the stomach by an esophageal tube lined with gastric mucosa. There was a concomitant aberrant artery arising from the subdiaphragmatic aorta. The esophageal communication of this case parallels the one just reported.

Baar and D'Abreu,[1] in 1949, reported the case of a 6-year-old child who was found to have an epiphrenic esophageal diverticulum attached to the posterior basal segment of the left lower lobe and connected to the esophagus by a patent lumen. The diverticulum was nourished by an aberrant systemic artery going to the cyst but not to the lung. The wall of this esophageal diverticulum contained pancreatic tissue, but there was no aberrant pancreas within the lung itself. Their case, to the author's knowledge, is the only other reported case of aberrant pancreas within the thorax.

Lynch and his associates[5] have recently reviewed the literature of the common types of intralobar sequestration and have highlighted the various theories as to embryologic origin. It is not the purpose of this article to explain the embryology of this unusual case. However, the finding of enteric tissue within what would otherwise be called a classical intralobar sequestration lends evidence to the theory that intralobar sequestration is an anomaly of foregut development and perhaps not the direct effect of the aberrant systemic vessel as proposed by Pryce.[6] It appears further to support Boyden's theory[2] that these anomalies are due to some diversion in the downward growth of the early lung bud. If such can be reasoned to occur, then it is also reasonable to expect that areas of the developing foregut, such as the stomach, esophagus, or even pancreas, can be captured within the developing lung bud.

SUMMARY

1. A case of intralobar sequestration of the lung with an unusual concomitant of aberrant pancreas and esophageal duplication is presented.

2. Successful management by lobectomy and division of the associated aberrant vessel and esophageal duplication is outlined.

3. This complex anomaly supports Boyden's theory as to the embryologic origin of intralobar sequestration.

The pediatric consultation of Dr. M. L. Levy and photomicrography by Dr. A. L. McQuown are gratefully acknowledged.

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THE OPERATIVE MANAGEMENT OF THORACIC AND THORACOABDOMINAL WOUNDS IN THE COMBAT ZONE IN KOREA

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AT THE ONSET of World War I, the Allies were prepared for the treatment of wounds as they were cared for during the Civil War, namely by dressing the wound and draining as necessary. They were unprepared for high velocity missiles, and the mortality from penetrating and perforating thoracic wounds was high, approximately 47 per cent for the American Forces (Table I).

TABLE I. MORTALITY FROM THORACIC WOUNDS

	THORACIC (PER CENT)	THORACOABDOMINAL (PER CENT)
World War I	47	–
World War II	9	27
Korea		
Rockey[2] (158 cases)	8	19
Artz[3] (29 cases)	–	10

During World War II the Allies were better equipped. Anesthesia had advanced, sulfanilamide was available, there was an increasing supply of whole blood, and penicillin was introduced. The mortality[1] for thoracic wounds was approximately 9 per cent and for thoracoabdominal wounds 27 per cent. At the onset of the Korean War, Mobile Army Surgical Hospitals replaced Field Hospitals as the most forward operating facilities, and Rockey[2] and Artz[3] reported on the initial salvage of the thoracic wounded.

This study is based on the management of 265 thoracic and thoracoabdominal missile wounds at the Mobile Army Surgical Hospital, 8055 Army Unit, in Korea between June 1, 1952, and March 31, 1953.

MATERIAL

Preoperative Resuscitation.—The hospital was situated on the relatively stable Western Front and the casualties arrived by ambulance and helicopter.

This study was made at the Mobile Army Surgical Hospital, 8055 Army Unit, Korea. The opinions expressed are the authors' and do not necessarily reflect those of the Medical Corps of the Army of the United States.

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An average of 5 hours elapsed between the time of wounding and arrival at the hospital. Morphine and plasma were often given en route. The majority (87 per cent) of the thoracic wounds were caused by shell fragments from mortar, artillery fire, hand grenades or land mines, and many patients had associated severe injuries. The wounds were penetrating in 190 cases (72 per cent) and perforating in 75 cases (28 per cent). There were 192 thoracic (73 per cent) and 73 thoracoabdominal (27 per cent) wounds.

During resuscitation, an attempt was made to restore cardiorespiratory balance, to evaluate the response to supportive treatment, and finally to consider the need for and extent of operative treatment. On admission all wounds were examined and sucking wounds of the chest wall were occluded by a petrolatum gauze dressing. The blood pressure, pulse, and respiratory rates were charted regularly. General supportive measures included nasal oxygen, Fowler's position, blood transfusion, and the relief of pain by narcotics or intercostal nerve block with 2 per cent procaine hydrochloride. Penicillin, one million units, and streptomycin, one gram, were given on admission and repeated by schedule during the hospital stay. Chest x-rays, posteroanterior and lateral, were made with the patient in sitting position, and air and fluid were removed from the pleural cavity by thoracentesis to allow re-expansion of collapsed lung. On occasion, tension pneumothorax was relieved by inserting a No. 14 Fr. catheter through the second intercostal space into the pleural cavity and connecting it to an underwater seal bottle. Flail chests were temporarily stabilized by sandbags. If tracheal or bronchoscopic aspiration was inadequate for the recovery of secretions, tracheotomy was performed. In suspected thoracoabdominal wounds, a Levin tube was passed into the stomach.

TABLE II. OPERATIVE MANAGEMENT

	EVACUATED	DIED	TOTAL
Débridement	147	5	152
Limited thoracotomy	85	2	87
Formal thoracotomy	22	4	26
Totals	254	11	265

Thoracic Wounds.—Table II summarizes the operative treatment and mortality rate of the 265 wounds in this series. One hundred and fifty-two patients required only simple débridement of the wound sites. Local or general anesthesia was used, depending on the extent of the tissue damage. Retained intrathoracic missiles were not considered adequate reason for further operative intervention. Repeated thoracentesis usually resulted in full expansion of the lungs and clear lung fields as judged radiologically. There were 5 deaths, 2 from the peritonitis of a thoracoabdominal wound and 3 from associated missile wounds.

A limited thoracotomy was performed upon 87 patients with more extensive damage to the chest wall. Usually there was a sucking wound and debris in the lung and pleural cavity. Under general anesthesia the chest wall, including

ribs, was débrided. Clotted blood, clothing, dirt, and readily accessible missiles were removed from the lung and pleural cavity. Traumatized lung tissue was minimally débrided and the pleural cavity was lavaged with saline solution. A dependent intercostal catheter connected to an underwater seal was often inserted and the débrided wound was closed in layers. There were 2 deaths from associated missile wounds in this group.

TABLE III. INDICATIONS FOR FORMAL THORACOTOMY

1. Uncontrolled intrathoracic bleeding
2. Uncontrolled tension pneumothorax
3. Destruction of the chest wall
4. Some thoracoabdominal wounds
5. Esophageal injuries
6. Cardiac tamponade

A formal thoracotomy was made in 26 cases, usually by an intercostal incision. The indications appear in Table III. In 11 patients there was intrathoracic bleeding from missile lacerations of the heart and great vessels which required an average blood replacement of 4,500 ml. Two instances of tension pneumothorax from laceration of the intrathoracic trachea were successfully repaired. Débridement of massive destruction of the chest wall in some cases constituted a formal thoracotomy. Tantalum gauze was used to help bridge the larger defects. A suspected diaphragmatic perforation was considered an indication for exploration, but thoracoabdominal wounds were approached primarily through the abdomen. At times the diagnosis of an esophageal laceration was difficult and in 2 patients the suspicion of esophageal laceration was not confirmed at exploration. Cardiac tamponade intractable to pericardial paracentesis was treated operatively on 5 occasions with the recovery of small missiles from the pericardium and heart muscle. There were 4 deaths during and following formal thoracotomy. These deaths were all related to the thoracic wound.

TABLE IV. THORACOABDOMINAL WOUNDS

	EVACUATED	DIED	TOTALS
Laparotomy	53	2	55
Formal thoracotomy	9	0	9
Limited thoracotomy	7	0	7
Thoracoabdominal incision	2	0	2
Totals	71	2	73

Thoracoabdominal Wounds.—The management of the 73 thoracoabdominal wounds is shown in Table IV. Most of these wounds were caused by shell fragments and frequently there were serious associated injuries. Consequently treatment demanded as little additional surgical and anesthetic trauma as possible. After correction of cardiopulmonary imbalance, most of the wounds were approached through the abdomen under general endotracheal anesthesia. Secondary repair of the thoracic wound was made by débridement or limited thoracotomy, depending on the extent of damage to the chest wall. In 9 cases of

diaphragmatic perforation there were frank indications for formal thoracotomy and in these the abdominal repair was made through the diaphragm from above. Seven wounds of the lower thoracic wall near the attachment of the diaphragm were treated by limited thoracotomy and the abdominal injury was adequately managed through the diaphragm. Two thoracoabdominal incisions were made to obtain adequate exposure for injuries in the cardioesophageal region. There were 2 deaths postoperatively, both directly related to the thoracoabdominal wound.

Postoperative Care.—The blood pressure, pulse, and respiratory rates were recorded regularly. Nasal oxygen, blood transfusion, and Levophed were given as indicated and the intake and output of fluids were charted. Penicillin and streptomycin were continued until evacuation. Chest roentgenograms were made in the sitting position and thoracentesis was carried out as indicated to empty the pleural cavity of fluid and air and keep the lung expanded. Coughing was encouraged and intercostal nerve block aided the patients in raising secretions. Tracheal and bronchoscopic aspirations were performed and, on occasion, tracheotomy was lifesaving. Flail chests were stabilized by towel-clip traction of a central rib for 3 to 4 days. The intercostal catheters were removed in 2 to 3 days and breathing exercises[4] were instituted early. Most patients were evacuated with the pleural cavity cleared of fluid, but with some linear atelectases, and resolving pulmonary hematomata.

COMMENTS

The proper management of thoracic war wounds has been gradually established. Each case, however, must be treated according to its own distinct features and in keeping with the local situation. Possible variables are the experience of the personnel, the number of casualties to be treated, the extent of associated missile injuries, the availability of surgical supplies, the evacuation facilities, and the tactical situation. The initial management of thoracic wounds is designed to restore cardiorespiratory balance, stop bleeding, and prevent infection. This is best achieved by giving as much attention to resuscitation as to the operative phase of wound management.

Resuscitation[5] is designed to return circulatory and respiratory function toward normal in 2 to 3 hours. Needless haste in operating may result in unnecessary mortality. Adequate resuscitation may eliminate the need for surgery. Failing this, it provides a background for the timing of surgical intervention, and improves the resistance of the patient to operative trauma. A good response to resuscitation is shown by an increase in the arterial blood pressure, a reduction in heart rate, and disappearance of the signs of peripheral vasoconstriction. When the findings suggest that the patient is not responding satisfactorily to treatment, although the thoracic condition is stabilized, immediate surgical intervention may be indicated. There may be hidden hemorrhage beneath the diaphragm or contamination from the gastrointestinal tract. Selection of the best time for intervention in these patients is difficult.

In patients with severe shock, the initial blood replacement was sometimes made intra-arterially and the response was dramatic. A readily available supply of blood required constant vigilance against overtransfusion and pulmonary edema, especially in hemothorax, pneumothorax, and pulmonary hematoma with a diminished pulmonary vascular bed. The severe extremes of temperatures to which casualties were exposed in summer and winter were considered in the management of fluid balance.

The technical aspect of operative management is concerned mainly with stopping bleeding, removing traumatized tissue and foreign bodies, and repairing defects. A major principle is to avoid unnecessarily long surgical procedures, especially in the patient with multiple wounds or associated major injuries. Unnecessary extension of the indicated procedures, such as tracking down questionably located missiles, should be avoided. Extensive débridement should be postponed 24 to 48 hours in casualties with multiple wounds who have undergone lengthy surgery for major thoracic injury.

In penetrating wounds requiring only débridement and thoracentesis there is nothing to be gained by the elective removal of retained missiles. However, on infrequent occasions the presence of a large shell fragment in a threatening position to the heart and great vessels, mediastinum, or diaphragm was a modifying consideration. With damage to the chest wall requiring limited thoracotomy, easily accessible missiles were removed.

Intercostal catheters with underwater seal drainage were not used to treat simple hemothorax. They were used freely in limited and formal thoracotomy and gave good postoperative drainage. No accidents occurred with their use and except on one occasion they were removed prior to evacuation of the patient from the hospital.

There were 11 missile wounds of the heart and great vessels with bleeding into the pericardial and pleural cavities with one death. Lacerations of the superior vena cava, inferior vena cava, right and left innominate veins, left subclavian vein, and the ventricular myocardium were treated by thoracotomy followed by recovery. The death was caused by a perforating bullet wound of the right auricle with uncontrolled hemorrhage and cardiac arrest during exploration. There were two penetrating missiles that migrated in the cardiovascular system. The first, a 2 by 1 cm. shell fragment, lacerated the right ventricle before lodging in the lumen of the intrathoracic inferior vena cava. A right thoracotomy was necessary to relieve cardiac tamponade. The shell fragment was dislodged at exploration and postoperatively was found by x-ray studies to have migrated to the left main pulmonary artery. The patient was a Republic of Korea infantryman. He was allowed to remain with the hospital and 2 months later the missile was removed through a left thoracotomy. In the second patient, a small shell fragment penetrated the chamber of the left ventricle producing pericardial and pleural hemorrhage and was found by x-ray examination to be lodged in the right femoral artery. The hemorrhage was controlled and the missile was recovered from the femoral artery.

Thoracoabdominal wounds, in general, required operative treatment more urgently than thoracic wounds. This was particularly true of left thoracoabdominal wounds with pleural and peritoneal contamination by large bowel contents or hidden peritoneal hemorrhage. These patients often did not make the expected recovery with adequate resuscitation and the timing of surgical intervention was difficult. Thoracoabdominal wounds were approached primarily through the abdomen, giving a more complete exploration of the peritoneal cavity than thoracotomy with exploration through the diaphragm. This was less traumatic than a thoracoabdominal incision with interruption of the costal arch and splitting of the diaphragm, and was an important consideration in patients with associated multiple shell fragment wounds requiring additional surgery. Most thoracic wounds required only débridement of the chest wall and this applied equally to the thoracic component of most thoracoabdominal wounds. At times the thoracic part of a thoracoabdominal wound constituted an indication for formal thoracotomy and in these patients the abdominal injury was repaired through the diaphragm.

There were 11 deaths (4 per cent) in this series. Five were due to associated missile injuries causing massive soft tissue damage. Three deaths were avoidable; 2 of these patients had left thoracoabdominal wounds with large and small bowel lacerations and were managed through an abdominal approach. A small laceration of the posterior antral stomach wall was overlooked in both patients. Postoperatively there was leakage from the stomach and death resulted from peritonitis. The third patient had a large shell fragment wound of the abdomen and underwent a right hemicolectomy with ileocolostomy. A thoracotomy was then made to recover another shell fragment lodged in the mediastinum. This exploration was poorly timed and the combined procedures proved too much; the patient died in pulmonary edema. There were three additional deaths, including the previously mentioned perforating bullet wound of the right auricle. One patient had a suspected shell fragment laceration of the lower third of the esophagus. The mediastinum was explored through the right chest and the laceration was located. At this point, aspiration of tracheal secretions abruptly resulted in cardiac arrest and resuscitation failed. In the third patient, massive destruction of the chest wall was repaired by formal thoracotomy. Postoperatively there was intractable arterial hypotension followed by death in 12 hours. At autopsy the cause of death was not determined.

The seriousness of the multiple wounds in these patients is emphasized by the fact that nearly one half the deaths were due to associated major injuries. There were only three deaths (1 per cent) among the 265 cases that were not due either to associated wounds or errors in treatment.

SUMMARY

The operative treatment of 265 thoracic and thoracoabdominal missile wounds at the 8055 Mobile Army Surgical Hospital in Korea from June 1, 1952, to March 31, 1953, is presented.

The timing and extent of the surgical procedures were determined after adequate preoperative resuscitation.

The high incidence of associated wounds accentuated the need to prevent any additional trauma from undue prolongation of surgery and anesthesia.

There were 11 deaths (4.7 per cent) including 2 deaths (2.7 per cent) from 73 thoracoabdominal wounds. Nearly one half the deaths were due to associated wounds.

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PULMONARY RESECTION WITH MECHANICAL SUTURE

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IN RESECTION of the lung, the question of the technique of bronchial suturing is still under discussion. In segmentectomies the method of treating the wound surface of the lung is as yet an unsolved problem. Although most surgeons prefer to leave it open, and confine themselves to the application of isolated ligatures on the injured vascular and bronchial branches as recommended by Overholt,[11] many still cover the wound surface with a pleural flap (Prochazka[12]), or carefully suture the entire surface (Cooley, Moser, and Hedberg[9]).

During the past 11 years (1949-1959), 2,744 resections of the lungs have been performed at the clinic of thoracic surgery at the Kiev Institute of Tuberculosis. In view of the central position of the Institute, operations at the clinic were performed on gravely ill patients who could not obtain surgical aid in the outlying districts. This circumstance had its effect on the extent of surgical intervention, and the percentage of such operations as pneumonectomy was as high as 43.5 per cent in tuberculosis and 53 per cent in suppurative diseases. The number and mortality rate of operations are given in Table I.

TABLE I

	NUMBER OF OPERATIONS	MORTALITY (PER CENT)
Tuberculosis	1,820	2.8
Suppurative diseases	757	4.5
Cancer of the lung	167	10.0
Totals	2,744	3.7

Operations for pulmonary tuberculosis predominated. The difficulties involved in these operations arose from the fact that surgical collapse methods of treatment are still fairly extensively used. Resection of the lungs was, therefore, performed chiefly in accordance with absolute indications, when surgical collapse proved ineffective or could not be used at all. Among the patients operated upon for tuberculosis, 86.1 per cent had positive sputum, and cavities were found in 97.2 per cent (in 40 per cent they were gigantic or multiple). Despite the gravity of these cases, complications were rare up to 1956—during the

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time when the patients seldom underwent treatment with antibacterial drugs, and drug-resistant bacteria were relatively rare—while the incidence of empyema and bronchial fistulas did not exceed 5 per cent. During 1956-1957 the results of pulmonary resection deteriorated because of the increase in the number of patients with tubercle bacilli resistant to antibiotics, and the incidence of complications rose to 8 per cent. This led to a search for new operative methods, to the extensive application of single-stage thoracoplasty and, finally, to a simplification of the lung resection procedure. It was also our aim to create an operation suitable for extensive resection of the lungs in hospitals of outlying districts.

It should be noted that the clinic of thoracic surgery is not only a central medical establishment but is also a base for the training of thoracic surgeons for the Republic. In the course of 4 years, about 100 thoracic surgeons were trained, and 70 of them performed 1,516 resections of the lungs in 1958, with an average mortality rate of 3.8 per cent.

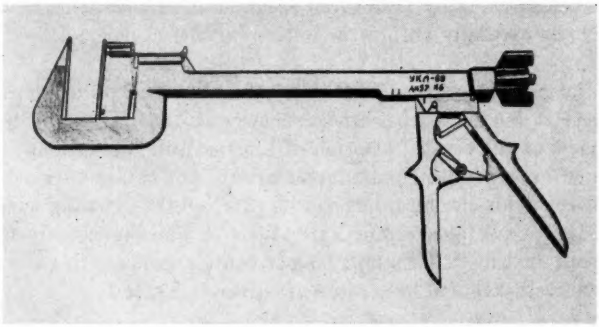


Fig. 1.—Lung root suturing device (UKL-60).

New opportunities for improving operative methods were opened up by the success of Soviet instrumental techniques and, in particular, by the development of the UKL-60 instrument for applying a mechanical suture by means of tantalum staples. The UKL-60 device was designed in 1957 at the Moscow Research Institute for Experimental Surgical Apparatus and Instruments by N. S. Gorkin and A. A. Strekopytov[5] for single-stage suturing of the lung root elements en masse. Its appearance is shown in Fig. 1. The length of the jaws is 60 mm. The device is loaded with 21 staples, 4.8 by 4 mm., in staggered order in two rows. The root of the lung or lobe is tightly clamped between the jaws, and suturing with the staples is effected by means of a single pressure of the handgrip, after which the part of the lung to be excised, or the entire lung, is cut off along the edge of the clamp. The operation lasts several minutes. The apparatus has successfully passed tests in our clinic, and has been highly appreciated in other large clinics of the Soviet Union.

For pneumonectomy we employ a procedure similar to that developed by E. S. Lushnikov,[10] in which the bronchus is first sutured high up at the bifurcation of the trachea, and the apparatus is then once more applied on all vessels

of the root of the lung. In upper lobectomies we first separate out and transect the segmental arteries, draw aside the main trunk of the pulmonary artery, and then suture all the elements of the lobe root with a single application of the device, so that a short bronchial stump is left. In lower lobectomies the root of the lobe may be sutured entirely.

The most advantageous effect was obtained on applying the UKL-60 device for partial resection of the lungs of two or three segments. A practically new operation was developed, the resection of the affected part of the lung being performed not along the segmental boundaries, but economically—along the boundaries of the focus of destruction. The apparatus is placed directly on the lung tissue near the focus of destruction (Fig. 2). The lung tissue is clamped by the jaws of the device and sutured with staples by pressing on the handgrip (Fig. 3). The part to be excised is cut off (Fig. 4) and a durable suture remains on the surface of the lobe which does not prevent it from expanding (Fig. 5).

In the course of 2 years (up to Nov. 1, 1959) 670 operations were performed with the UKL-60. Table II gives the type of operation, the incidence of bronchial fistulas, and the mortality.

In pneumonectomies and lobectomies, a two-stage treatment of the lung with the aid of UKL-60 permits removal of the lymphatic nodes and a fairly high excision of the bronchus. The stumps of vessels outside the staples may be utilized for covering the bronchus stump with tissues. Defects of the mechanical suture in the form of a slight bleeding occurred in 11 patients. The defects were easily eliminated by one or two additional sutures. Bronchial fistulas and empyemas of the pleural cavity arose in 10 gravely ill patients. Two of these patients died, and 4 died of cardiac insufficiency.

Partial resection with dissection of lung tissue was performed in 340 cases with the UKL-60 device. Of particular interest are economical resections with the UKL-60 in tuberculosis; these yielded far better results than typical segmentectomies. Of 206 operations, 178 were performed for cavities, 23 for tuberculomas, 5 for caseous foci. Sixty-eight per cent of these patients had positive sputum. Many of them had undergone prolonged but unsystematic antibiotic treatment; 96 had been treated with collapse without effect.

Variations of the methods of partial resection depend on the localization and dimensions of the affected areas. Apical resections (Fig. 6, *a*) were performed on 118 patients for lesion of the first and sixth segments in cavities or tuberculomas of peripheral localization. Wedge resections (Fig. 6, *b*) were performed in 62 cases, when the cavity was located deep in the lobe tissue. At the beginning, mechanical suturing was applied at a tangent to the cavern. After suturing along this line the affected part was drawn aside and cut off in the second stage, completing the wedge excision. In this double complicated suture, the weak point was the contact of the two lines, where an additional mattress suture was often required. A draining bronchus must not get into the angle between the lines. When the cavities are deep in the root, the segmental bronchus is first separated out and transected by the ordinary procedure, thus avoiding coarctation of the bronchial and vascular elements of the remaining segments.

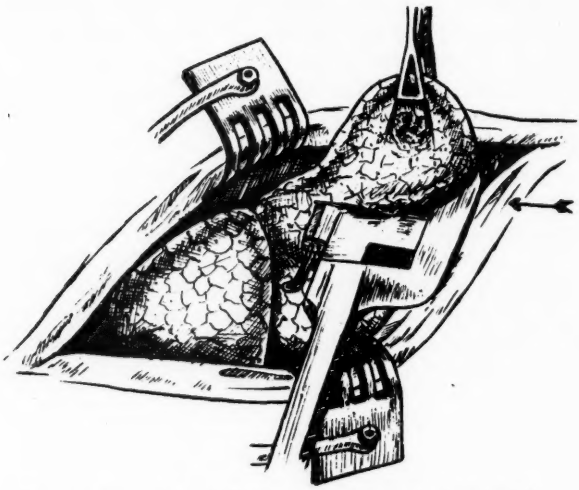


Fig. 2.—Placing the UKL-60 on affected part of upper lobe.

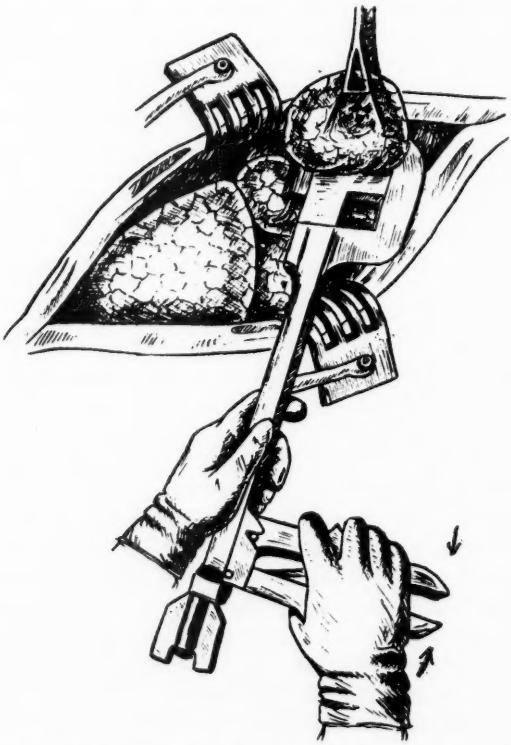


Fig. 3.—Suturing lung tissue with the UKL-60 device.

Polysegmental economical resections were performed on 27 patients with adjoining segments of various lobes affected, most frequently the second and the sixth. The affected segments are removed in one block. For this purpose a tunnel is made in the root area of the interlobar fissure through which the lower jaw of the apparatus is inserted, and the affected segments are then resected in turn.

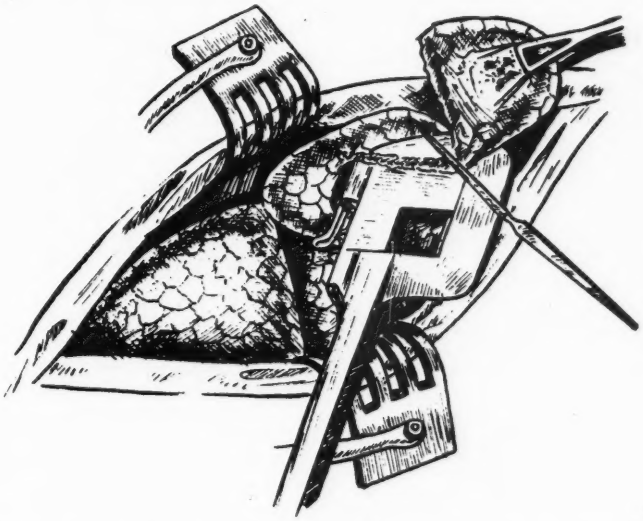


Fig. 4.—Cutting off affected part of lobe after suturing with the UKL-60.

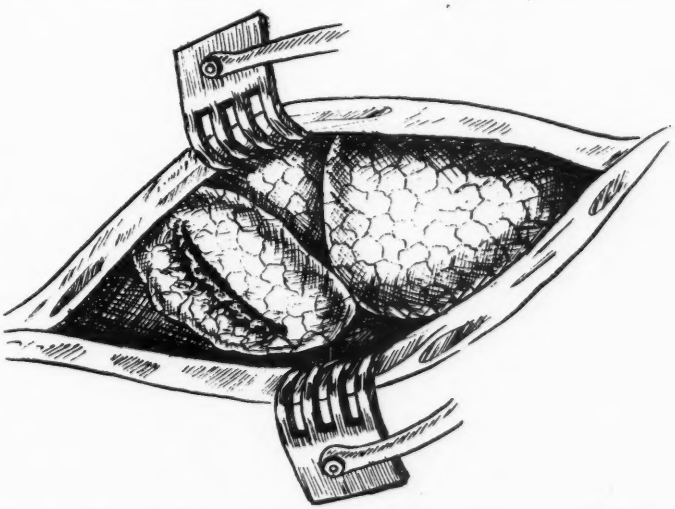


Fig. 5.—Appearance of mechanical suture of lung tissue after expansion of lung.

TABLE II

	TUBERCULOSIS			SUPPURATIVE DISEASES			CANCER OF THE LUNGS			TOTAL OPERATIONS
	TOTAL OPERATIONS	BRONCHIAL FISTULAS	DEATHS	TOTAL OPERATIONS	BRONCHIAL FISTULAS	DEATHS	TOTAL OPERATIONS	BRONCHIAL FISTULAS	DEATHS	
Pneumonectomies	91	6	4	36	—	—	51	2	2	178
Lobectomies	97	4	1	50	—	—	5	—	—	152
Economical resections within one or two segments	206	2	1	20	—	—	—	—	—	226
Excision of lobe and segment	32	6	1	6	—	—	—	—	—	38
Resection in one-stage bilateral operations	44	2	1	32	2	—	—	—	—	76*
Totals	470	20	8	144	2	—	56	2	2	670

*Seventy-six operations performed on 38 patients.

Flat resections (Fig. 6, *c*) are performed for the removal of small subpleural foci in conjunction with other partial resections.

In the absence of adhesions, intrapleural manipulations take a few minutes only, and the length of the operation depends on the time required for the incision and suture of the thoracic wall. After partial resections, the lung wound remains completely sealed, the lung expands well and fills the pleural cavity at once. Hemorrhage is insignificant and, in the absence of intrapleural adhesions, the loss of blood does not exceed 300 ml. No pleurization is performed. The bronchial stump should be submerged in tissue only in the case of a two-stage resection.

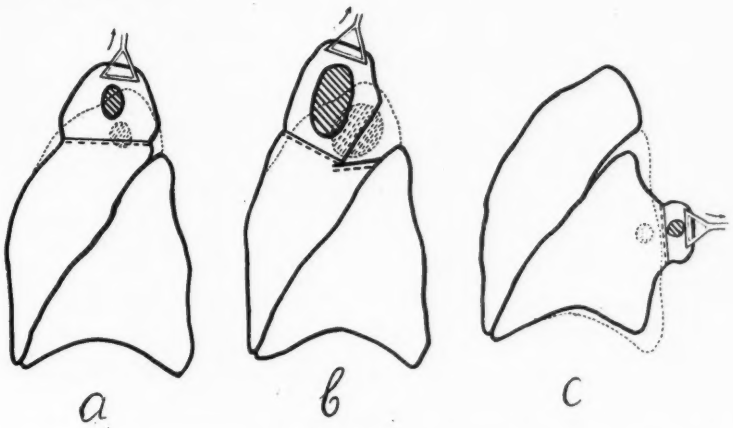


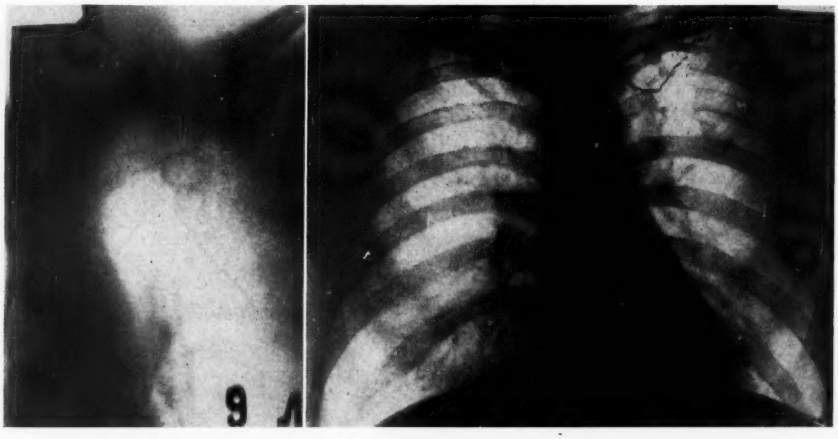
Fig. 6.—Variations of economical resection of lung with the UKL-60, *a*, Apical resection; *b*, wedge resection; *c*, flat resection.

Complications are rarely encountered during the operations. Air leaks from the lung wound, which required additional sutures, were observed in 9 cases. Slight hemorrhage occurred in 17 cases, and was arrested by swabs or sutures. The occurrence of suture defects depends on the varying thickness of the resected parts of the lung which require different degrees of clamping. Twenty-six patients after prolonged collapse treatment, in whom the lung failed to expand sufficiently during the operation, as well as patients with focal changes in the lungs, were subjected to limited thoracoplasty along with resection. Recently, filling with spongy plastic material (Porolon) was successfully performed simultaneously with resections in 18 patients in order to fill the residual cavity.

Retarded expansion of the lung requiring slight thoracoplasties was noted during the postoperative period in 8 patients; a pulmonary-pleural fistula being sutured in one of these cases. Suppuration in the pleural cavity with a bronchial fistula developed in 1 patient who had undergone combined resection for a 4-year-old gigantic cavity and who had displayed resistance to antibiotics. A supplementary thoracoplasty was performed, but the tuberculous process in the lung progressed and the patient died after 8 months.

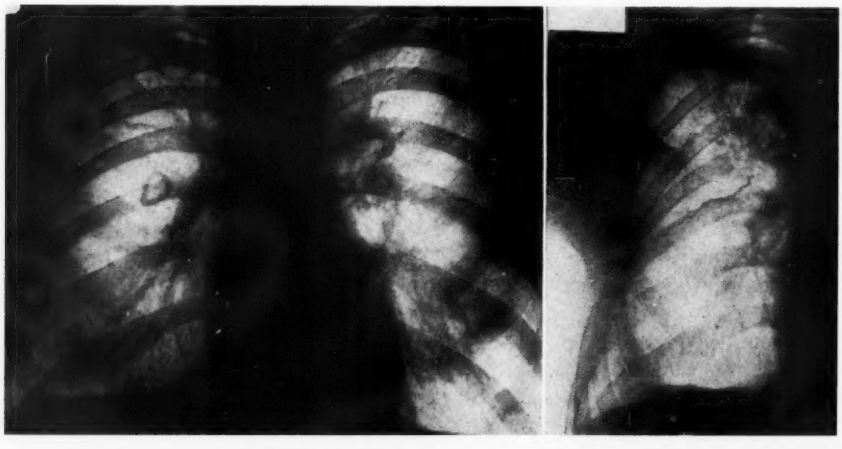
After being discharged from the clinic, patients are under observation from 2 months to 2 years. No inflammation was observed around the staples during this period. There were no complications in connection with the remaining length of the segmental bronchial stump. Radiographs of the lungs after partial resections with mechanical suture are presented in Figs. 7 and 8.

Economical resections with UKL-60 in suppurative diseases also confirmed the advantage of the new methods. Such operations were performed on 20 patients and no complications resulted.



A. *B.*

Fig. 7.—*A*, Tomogram of left lung of patient S. *B*, Radiograph of lungs of same patient after resection of apical segment of left lung.



A. *B.*

Fig. 8.—*A*, Radiograph of lungs of patient F. Cavities in 1 - 2 and in 6 segment of right lung. *B*, Radiograph of right lung after resection of 1 - 2 and 6 segments with the UKL-60. Space above clavicle is filled with Porolon.

The postoperative period was less favorable after large combined resections —removal of the upper lobe or two lobes (on the right) and the sixth segment. Leaving only the basal segments frequently leads to the complications of empyema and bronchial fistula; nevertheless, such complications are encountered less often with the use of the UKL-60 device in these operations, especially when performing thoracoplasty or filling of the residual cavity at the same time.

Bilateral one-stage operations are performed by two groups of surgeons simultaneously. As in other operations, intubation anesthesia with relaxants is employed. The operative approach is most often dorsolateral and less frequently anterior. A total of 44 one-stage bilateral operations were performed.

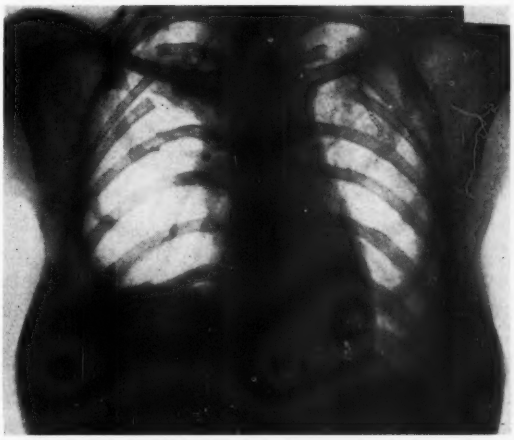


Fig. 9.—Radiograph of lungs 1 month after bilateral one-stage partial resection.

Of these, 38 were performed employing the UKL-60 device, with one lethal outcome. The observations of I. P. Dedkov[4] showed that application of the UKL-60 device reduced hemorrhage to a minimum and decreased the operating time to 1½ hour and 20 minutes. Complete sealing of the sutures helps the rapid expansion of the remaining parts of the lungs and prevents the development of respiratory insufficiency. Although 5 to 12 segments were removed on the average from both sides, only in 10 cases did it become necessary to resort to tracheotomy for improvement of sputum evacuation and oxygen therapy. Fig. 9 presents a radiograph of the lungs taken 1 month after a bilateral one-stage resection with the UKL-60.

DISCUSSION

The application of the UKL-60 stapling device simplifies resections of the lungs and permits performing them with less risk.

The advantages of mechanical suture in pneumonectomies and lobectomies consist in the rapidity and simplicity of the operation, but the incidence of

bronchial fistulas remains about the same as with the ordinary procedure. This shows that the cause is a specific lesion of the bronchi (tuberculous endobronchitis). A typical feature is that in resections for suppurative disease there are almost no complications, since the bronchi are affected to a lesser extent, and the processes of healing are less disturbed. Two years of observation of patients operated upon for cancer of the lung showed that the number of early relapses did not increase.

Partial resections can be performed with the aid of the UKL-60 by the principle of excision of the affected part of the lung. The basic method consists of mechanical suture of the lung tissue, which creates complete sealing and good hemostasis along the line of transection. An important feature of the method is that the lung tissue is first sutured along the boundaries of the lesion in one row, and the affected part is cut off, i.e., a closed line of resection is formed, which reduces to a minimum the danger of infecting the pleural cavity. The zone of resection almost always involves the peripheral part of adjoining segments, since the plane of resection does not coincide with the intersegmental boundaries. This permits calling such operations regional resections, i.e., performed in the region of definite segments.

The healing of lung tissue in the region of the tantalum staple suture was verified in an experiment on animals. We were convinced that tantalum gives rise to a minimum reaction of the lung tissue, while the union of the bronchial stump walls is well defined by the eighth to tenth day. Morphologic investigations (A. F. Fedotov) of the resected parts of the lung showed that in 60 per cent of the patients active changes occur in the bronchus on the line of incision; however, that did not affect the outcome of the operations.

Remote observations show that the line of mechanical suture of the lung tissue gradually extends, and by the end of the month the staples are aligned and subsequently small spaces appear between them. Thus the suture only slightly limits the expansion of the lung tissue.

On resecting lungs for tuberculosis with the aim of preventing complications and reactivation of the process we take measures to reduce the pleural cavity. The indications follow: drug resistance of the bacteria, foci in the remaining parts of the lung, poor expansion of the lung after prolonged collapse treatment, and after resections of large bulk. These indications were followed in lung resections with the UKL-60. Limited osteoplastic thoracoplasty with attachment of the peripheral costal segments to the intervertebral ligaments was performed with resection in 56 of 470 patients. The residual cavity was filled with Porolon in 42 patients. The experimental and clinical investigations of A. G. Gubanov, G. G. Gorovenko, and the authors showed that the surrounding tissues quickly grow into the Porolon cell, and the cavity is obliterated. There were no complications after filling.

Employing the UKL-60 device in bilateral one-stage partial resections enabled us to extend the indications for these operations and make them comparatively safe. In these operations the filling of the residual cavity is particularly promising, since it excludes the necessity for thoracoplasty.

The extreme simplification of operations with the UKL-60, yielding good immediate and long-term results, allows us to hope that the new resection procedure will find extensive application in lung surgery. At present, resections with the UKL-60 are being successfully performed at eight regional dispensaries of the Ukraine, where 326 operations have been performed with good results.

Partial resections may be performed with minimum risk. They are incomparably less traumatic than segmentectomies, and may replace them fully with better results.

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USE OF INTACT LUNG FOR CLOSURE OF FULL-THICKNESS ESOPHAGEAL DEFECTS

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THE REINFORCEMENT of questionable esophageal anastomoses and the closure of traumatic and spontaneous esophageal perforations are of genuine clinical interest. A series of investigations have been carried out recently to evaluate the suitability of various means of reinforcement and closure. The adequacy of the closure of a relatively large full-thickness esophageal defect of standard size has provided the yardstick for comparing the suitability of the various materials and methods. The purpose of this report is to record the experience with the experimental closure of relatively large full-thickness esophageal defects with intact lung.

MATERIAL AND METHODS

Healthy mongrel dogs were used. An effort was made to limit the oral intake of liquids and solid foods for a short period prior to operation. Intravenous Pentothal was utilized for anesthesia. Oxygen was administered through an endotracheal tube by means of a mechanical respirator. The left pleural cavity was entered through an intercostal space incision. The area of the junction of the middle and lower thirds of the esophagus was mobilized and secured between Potts coarctation clamps. An oval full-thickness defect of standard size, measuring approximately 2.5 by 2 cm., was made by resecting a segment of esophageal wall (Fig. 1). The Potts coarctation clamps were useful not only for manipulation of the esophagus, but also in the control of bleeding along the margins of the defect and the prevention of possible contamination of the pleural cavity and the area of operation by gastric and upper esophageal contents.

An intact segment of adjacent lung was used to close the defect in the esophagus. The segment of lung used generally was the upper portion of the lower lobe. The adjacent lung was secured to the margins of the esophageal defect by means of a series of 5-0 arterial silk sutures. Two types of suture closure were employed. In the first group of 16 dogs, interrupted sutures in

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2 or 3 layers were employed to secure the adjacent lung to the margins of the defect. In the second group of 19 animals, an inner continuous layer of suture for water-tight closure was reinforced by a second layer of interrupted sutures.

After completion of the closure, the thoracotomy incision was closed in layers and a catheter which was left in place during the closure was aspirated and withdrawn. The animals were given nothing by mouth for the first 24 hours after operation. On occasion, intravenous fluids were given during this period. For the following 10 days they were given milk by mouth. A soft meat diet then was given for one month prior to the resumption of a regular diet. Intramuscular penicillin, 800,000 units a day, was given for the first 5 days after operation.

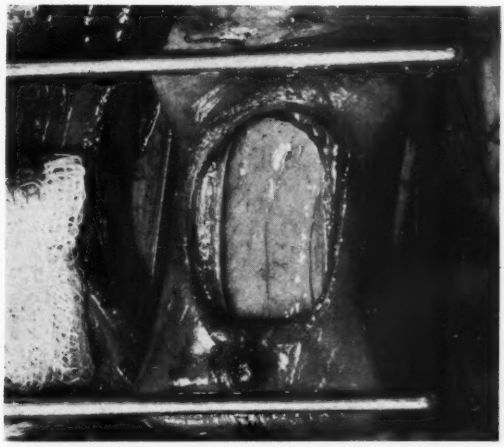

Fig. 1.—Photograph showing the appearance of the standard sized full-thickness esophageal defect employed in this study. The mobilized esophagus has been occluded above and below the defect with noncrushing Potts coarctation clamps.

RESULTS

The closure of full-thickness esophageal defects of standard size with intact adjacent lung was carried out as described in 35 dogs.

Defect closure with adjacent lung was carried out with interrupted sutures in 2 or 3 layers in 16 animals. Successful defect closure was achieved in 12 of the animals. Five animals died and 11 were sacrificed. The deaths occurred from 2 to 10 days following operation and 4 of the 5 deaths resulted from leakage of esophageal contents into the left pleural cavity from the area of defect closure. These were regarded as defect closure failures. One of these animals had been fed meat and solid foods shortly after operation by mistake. Two animals were sacrificed 6 weeks and 6 months after operation. The remaining 9 dogs were sacrificed 7 and 8 months following operation. Good defect closure and healing had occurred in all of these animals.

The defect in 19 dogs was closed with 2 layers of sutures, the inner layer being a continuous one. Successful closure was obtained in 17 of the animals.

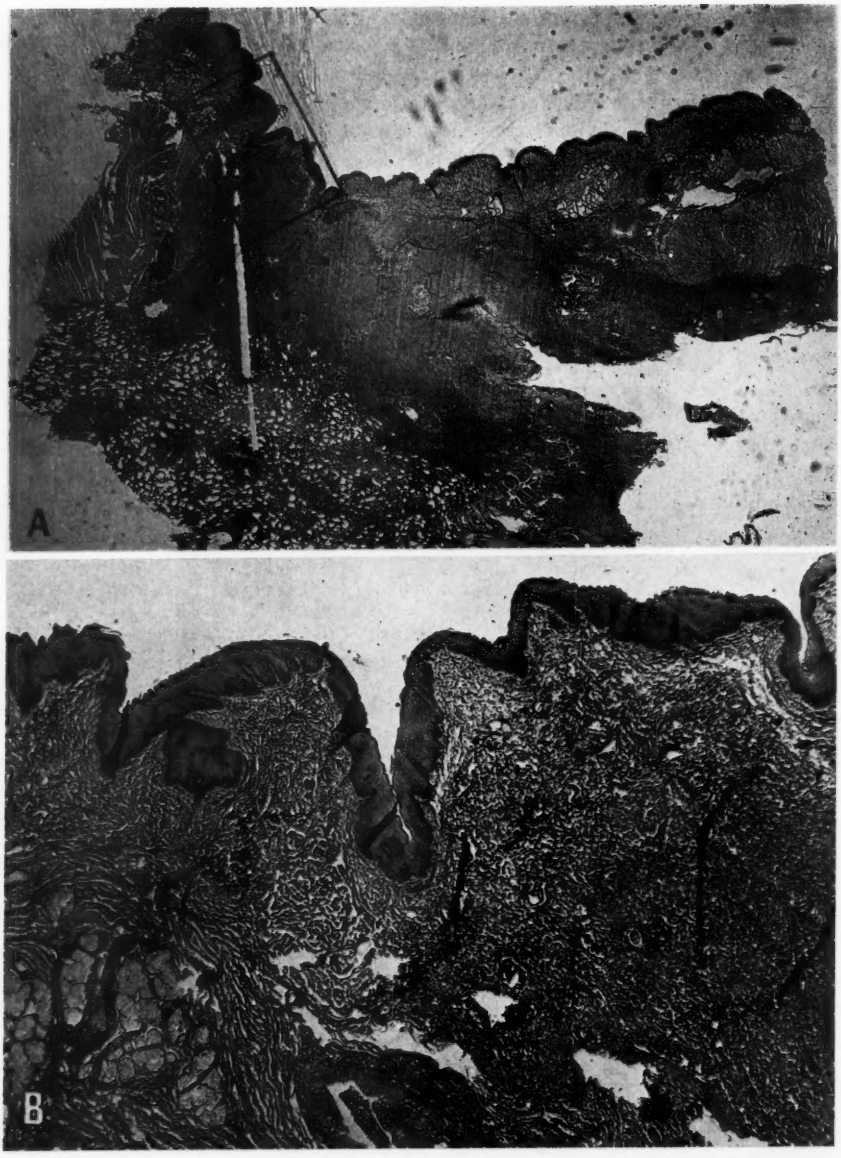

Fig. 2.—Photomicrographs showing the area of defect closure under low power *(A)* and somewhat higher power *(B)* in an animal sacrificed 7 months after operation. The higher power view presented in *(B)* is the area enclosed by the rectangle in the upper left corner of *(A)*. The scar tissue between the lung parenchyma and the lumen of the esophagus is covered by a layer of squamous epithelium.

Five animals died, 13 were sacrificed, and 1 disappeared from the colony after having survived a successful defect closure. The deaths occurred from 2 to 16 days after operation. All of the sacrifices were carried out 2 and 3 months following operation. The 2 deaths due to leakage in the area of defect closure occurred in the initial group of 8 animals in which this type of repair was performed. Rather vigorous attempts to re-expand the lungs fully after thoracotomy closure by positive pressure through the endotracheal tube were employed in this initial group. Subsequent investigation of this method of expanding the lungs with the chest open following this operation demonstrated that there was a tendency for some of the securing sutures to pull out of the overexpanded lung in a manner which tended to compromise the adequacy of the closure and to produce varying degrees of laceration to the pulmonary

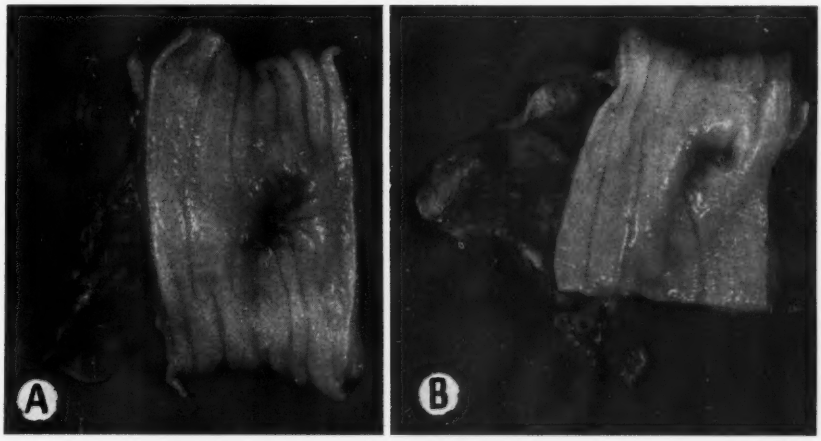

Fig. 3.—Photographs showing the area of esophageal defect closure in 2 animals sacrificed 3 months following operation.

parenchyma. Greater care was exercised in re-expanding the lungs in subsequent operations, largely through suction applied to the catheter left in the chest during thoracotomy. Defect closure was successful in all of the 11 operations carried out following this alteration in the technique of lung re-expansion. It was of interest that a rather large esophageal tumor was encountered in 1 of these animals. It was necessary to create a defect somewhat larger than the standard size in this animal to excise the tumor completely. The tumor was found to be a large granuloma which contained a worm. The esophageal defect in this animal was closed successfully with intact adjacent lung.

The healing in the area of defect closure followed a rather constant pattern in all of the animals. The plug of lung which closed the defect resembled granulation tissue and became covered with squamous epithelium (Fig. 2). Some contraction of the defect closure area occurred with the passage of time. The closure area often was relatively large even at 3 months following operation (Fig. 3). However, by 8 months after operation, the defect closure area

generally had become rather contracted with the formation of an irregular linear type of scar (Fig. 4).

In none of the animals in this study was narrowing or stricture formation at the site of defect closure observed. Neither esophageal dilatation nor diverticulum formation occurred in the area of closure.

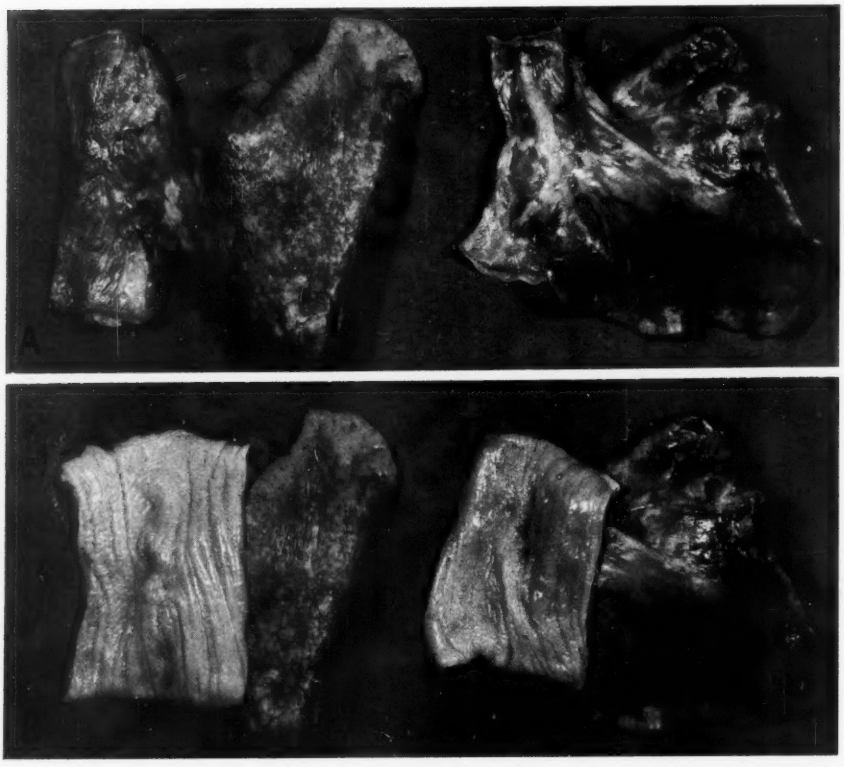

Fig. 4.—Photographs of the area of esophageal defect closure *(A)* before and *(B)* after opening the esophagus in 2 animals sacrificed 8 months after operation. The area of defect closure in the opened esophagus appears as an irregular linear scar.

DISCUSSION

Prior experience with the use of intact omentum and of nylon-mesh reinforced free pericardial grafts for closure of standard sized full-thickness esophageal defects has been reported elsewhere.[1, 2, 3] Intact omentum brought into the chest through a hole in the diaphragm was employed successfully for defect closure in 90 per cent of 31 "good test of operation" dogs.[1] Defect closure with nylon-mesh reinforced free pericardial grafts was satisfactory in 88 per cent of 24 animals.[2]

The clinical use of lung for esophageal support was reported in 1949 by Nissen.[4] He described the use of an intact pedicle flap of pulmonary parenchyma to support an area of circumferentially weakened esophagus. A rather

sizable area of esophagus had been weakened by the circumferential extramucosal excision of an extensive encircling leiomyoma of the esophagus. Although the lumen had not been entered, a segment of esophagus 10 cm. long, was denuded. The patient survived.

In this study, intact adjacent lung was used to close a standard size full-thickness esophageal defect in 35 dogs. Successful closure of the defect was achieved in 12 of 16 dogs in which interrupted sutures in 2 or 3 layers were employed to secure the adjacent lung to the margins of the defect. A 2 layer closure in which an inner continuous layer was employed for defect closure was used in 19 animals. Defect closure was successful in 17 of the 19 animals. A technical error relating to the method of pulmonary re-expansion following chest closure was suspected as being responsible for the 2 failures in this group. Successful defect closure was achieved in 11 consecutive animals by this method of closure following alteration in the technique of lung re-expansion.

The results of this study suggest the suitability of intact adjacent lung for the experimental closure of relatively large full-thickness esophageal defects. The use of a continuous, water-tight inner layer to secure the lung to the margins of the defect reinforced by a layer of interrupted sutures appears to be slightly superior to the use of interrupted sutures only in 2 or 3 layers. The use of intact adjacent lung for the reinforcement of questionable esophageal anastomoses and for the closure of traumatic and spontaneous perforations appears to be worthy of clinical trial when indicated.

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USE OF A LOBE OF THE LUNG TO SECURE SUTURES OF THE ESOPHAGUS

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THE greatest problem in surgery of the esophagus is dehiscence of the suture line. As the esophagus has no serous membrane, the suture line cannot be protected with it, as in abdominal anastomoses. Sutures of the thoracic esophagus are, therefore, especially endangered, and their insufficiency may cause serious pleural infection, often with a fatal outcome. The problem was taken to the experimental laboratory, where, in a series of experiments, injuries

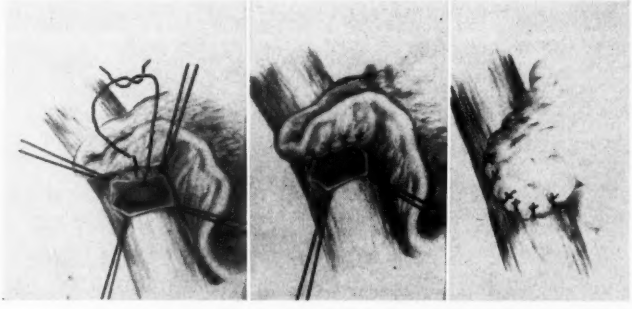

Fig. 1.—Drawing which shows the covering of an experimental defect of esophagus with lung.

of various degrees were inflicted that penetrated into the lumen of the thoracic esophagus of dogs; the wound was then repaired in one layer with interrupted sutures. All dogs died of a purulent pleuritis. The same operation was performed in a second series of dogs in which the suture line was covered by wrapping it with lung. In this group all dogs survived. In another series, the injury penetrating into the lumen of the thoracic esophagus was not sutured, but the lung was circularly stitched to the edges of the wound. These animals also survived without exception (Figs. 1-4).

In another series, a portion of the thoracic esophagus, 4 to 5 cm. long, was resected, the circular defect encircled with the lung as a muff, and the transsected esophageal edges were sutured to it. In 2 out of 5 dogs the experiment was successful, and no stenosis developed. Three dogs perished 24 hours after operation because of a bilateral pneumothorax.

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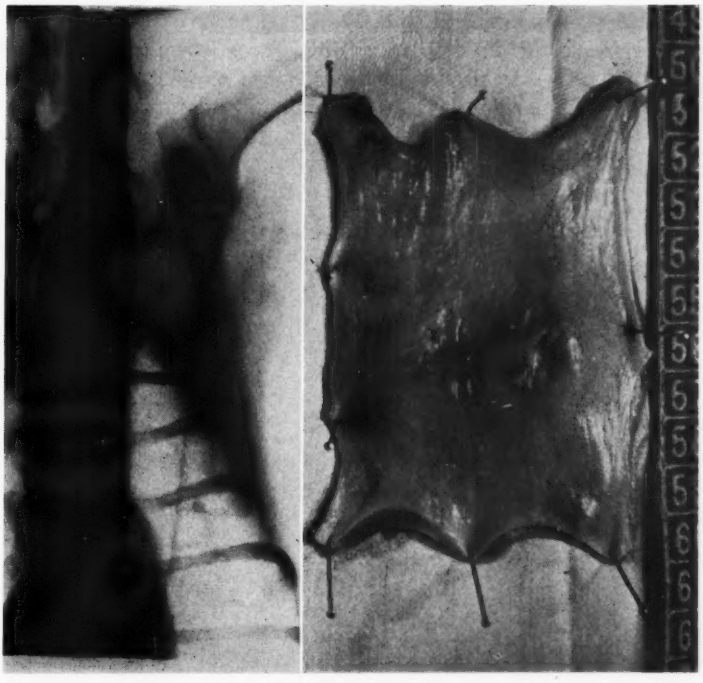

Fig. 2. Fig. 3.

Fig. 2.—Roentgenogram of the esophagus of a dog, 6 weeks after covering an esophageal defect with lung.

Fig. 3.—Interior view of esophagus 4 months after closing an esophageal defect with lung.

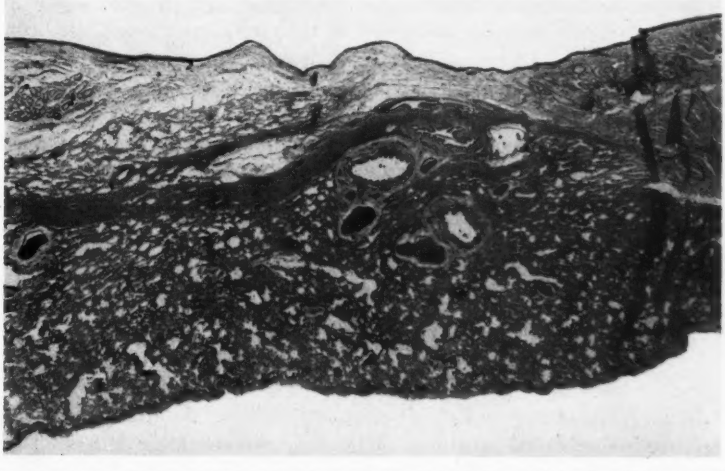

Fig. 4.—Microscopic appearance of the esophageal defect covered by lung shown in Fig. 3. The surface is covered everywhere by hornified epithelium. The layers of the esaphagus—tunica propria, muscularis mucosae, salivary gland, and the layers of the musculature—are observable on both sides. The lung affected by atelectasis lies immediately behind the epithelium corresponding to the scar.

Swallowing was undisturbed in the dogs that survived; no infection of the lung was observed at the place of the suture. X-ray examinations showed only occasionally the place of the circumscribed defect covered with lung; in the case of the circular muff-plasty, an insignificant dilatation was observable, corresponding with the site of the supplement. There was no peristalsis in this portion. After 3 to 6 months the experimental animals were sacrificed. Histologic examination showed that at the site of the defects the wall of the esophagus formed only by the tissue of the lung affected by atelectasis and by a stratiform epithelium above it. The muscularis mucosae, the salivary glands, and the muscular layer were missing. (Figs. 5-8).

The experiments verified that the lobe of the lung sewed over a suture line prevents dehiscence and, because of this, the purulent pleuritis.

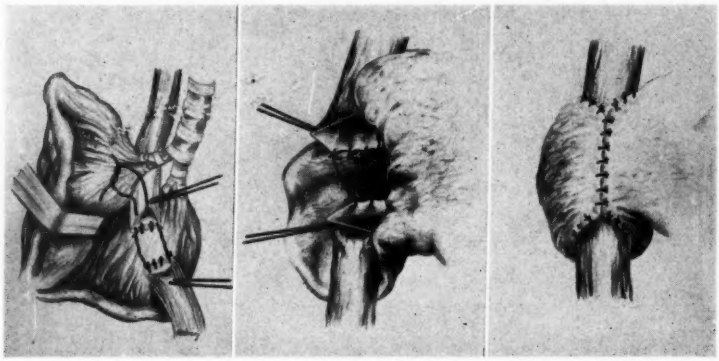

Fig. 5.—Drawing which shows the replacement of an esophageal defect with lung.

The experimental observations were applied in clinical cases. In 10 instances (7 tumors of the cardia, 2 tumors of the esophagus, 1 esophagobronchial fistula), the suture line in the esophagus was secured by covering it with a lobe of the lung, anchored by interrupted silk stitches. In these cases, no insufficiency of sutures occurred.

In 2 other cases, a tumor of the esophagus, and one of the cardia, the sutures of the esophagus were not secured with the lung. On the fourth to fifth postoperative day, the evidence of suture insufficiency was noted. After establishing continuous thoracic suction, the lung adhered to the site of the insufficiency and both patients recovered (Table I).

On the basis of these experiences the author suggests the securing of an esophageal suture line by covering it with lung.

SUMMARY

1. In the series of experiments on dogs, injuries and defects of the esophagus were covered or replaced by lung. The dogs survived, no infection of the pleura occurred, and no stenosis developed.

2. Histologic examination showed that the scarred surfaces were covered by regular hornified epithelium under which only the tissue of the atelectatic

lung was observable. The other layers of the esophagus (the tunica propria, the muscularis mucosae, the salivary glands, and the two layers of the deep musculature) were missing.

Fig. 6.

Fig. 7.

Fig. 6.—Roentgenogram taken 5 weeks after circular resection which shows no stenosis.

Fig. 7.—The incised esophagus of dog sacrificed 2½ months after replacing esophageal segment by lung.

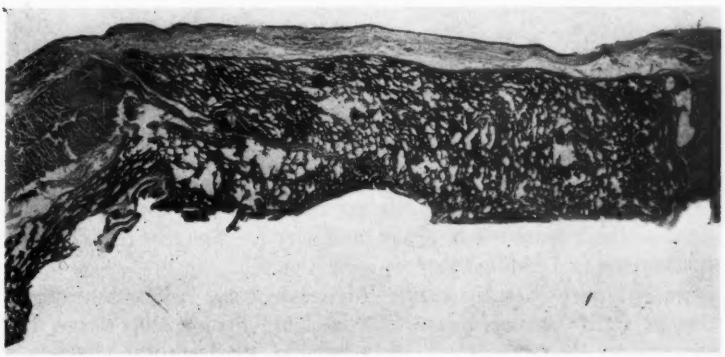

Fig. 8.—Microscopic appearance of esophagus shown in Fig. 7.

TABLE I

NO.	NAME	AGE	SEX	DIAGNOSIS	OPERATION	SUTURE OF LUNG	INSUFFICIENCY OF LUNG	NOTE
1	J. K.	68	M	Fistula, esophago-bronchial	Extirpation + suture	+	–	
2	J. T.	56	M	Tumor of cardia	Resection + esophago-jejunostomy	+	–	
3	M. L.	68	M	Tumor of cardia	Resection + esophago-gastrostomy	+	–	
4	J. S.	67	M	Tumor of cardia	Resection + esophago-gastrostomy	+	–	
5	F. O.	66	M	Tumor of cardia	Resection + esophago-gastrostomy	+	–	
6	M. H.	56	M	Tumor of cardia	Resection + esophago-gastrostomy	+	–	
7	I. A.	53	M	Tumor of cardia	Resection + esophago-jejunostomy	+	–	
8	P. P.	53	F	Tumor of esophagus	Resection + esophago-gastrostomy	+	–	
9	I. R.	63	M	Tumor of cardia	Resection + esophago-gastrostomy	+	–	
10	A. K.	54	F	Tumor of esophagus	Resection + esophago-gastrostomy	+	–	
A	P. R.	67	M	Tumor of esophagus	Resection + esophago-gastrostomy	–	+	Permanent suction corrected the insufficiency
B	P. M.	55	F	Tumor of cardia	Resection + esophago-gastrostomy	–	+	Permanent suction corrected the insufficiency

3. In the course of ten surgical interventions on the esophagus, the author secured the suture line by stitching the lung over it. The sutures were safe, and the patients recovered without any complication.

In two instances when the suture line was not secured by the lung, an insufficiency of suture occurred on the fourth and fifth postoperative days, respectively. Through continuous thoracic suction, the lung was made to adhere to the site of the insufficiency and both patients recovered.

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THE USE OF DIAPHRAGM GRAFTS FOR PLASTIC OPERATIONS IN THORACIC SURGERY

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At the Leningrad Oncological Conference in 1948, we suggested using the diaphragm in plastic operations on the esophagus. Later, this method was elaborated upon in clinical and experimental work by E. N. Vantsyan, T. A. Suvorova, O. D. Fyodorova, G. A. Natsvlishvili, I. Z. Kozlov, and others. The diaphragm is uniquely resistant to necrosis, is strong, elastic, and has a good regenerative quality. It has a serous lining on both sides and is well supplied with nerves and blood. The innervation of the diaphragm is effected through the branches of the phrenic and vagus nerves, six intercostal nerves, and the diaphragmatic plexus, the latter being a continuation of the solar plexus. The blood supply of the diaphragm is drawn from the pericardiophrenic arteries, the internal mammary artery on both sides, the inferior phrenic arteries (branches of the abdominal aorta), the six lower intercostal vessels, and partially from the gastric vessels. T. A. Suvorova described a wide intravisceral network of the lower diaphragmatic arteries. It should be pointed out that as a constantly functioning and rhythmically contracting muscular organ, the diaphragm can only be compared with the heart.

All these factors, together with the possibility of cutting out large grafts of the diaphragm with the subsequent closing of the defects without tension, prompted us to use these grafts for plastic intrathoracic operations.

In Budapest in 1948-1950 we studied closing defects in the esophagus, wounds of the liver, heart, and lung. We tested grafts fixed in place on pedicles of different lengths as well as free diaphragmatic grafts. These experiments demonstrated very good implantation of the grafts into different organs. Grafts affixed to pedicles and cut with consideration for innervation and blood supply grew well and preserved their structure for considerable time. The vascular collaterals of the graft became connected with the vessels of the organ. Free, loose grafts grew, but became wrinkled and underwent scarring. Two successful cases of diaphragmatic grafting in operations on the esophagus were described.

At the present time diaphragmatic grafts are used (1) for the correction of defects of the wall, or strengthening the line of anastomosis in operations on

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the esophagus, (2) for plastic operations on the cardia in cases of cardiospasm, (3) for the creation of artificial cardia in cases of gastroesophageal regurgitation, (4) for closing wounds in the lung, liver, heart, and aorta incurred during operations on these organs, and (5) for plastic operations upon aneurysm of the heart and for improvement of the myocardial blood supply.

In resection of the esophagus and cardia and in cases of gastrectomy we often face the necessity of strengthening the anastomosis of the esophagus when there is tension on the suture line or when the blood supply to the wall of the esophagus or adjacent organ, such as stomach or jejunum, is impaired. By using grafts from the diaphragm for covering the anterior or posterior lines

Fig. 1.—Patient B. Leiomyoma of the esophagus. Roentgenogram made prior to surgery.

of the anastomosis, or for completely wrapping it, not a single opening of the gastroesophageal or gastrointestinal anastomosis was observed in 15 patients in whom we performed this operation. Neither was there any narrowing of the anastomosis suggesting scarring and fibrosis of the graft.

Diaphragmatic grafts are also applicable in operations for benign lesions such as leiomyomas, cysts, and diverticula of the esophagus. In 10 patients grafts were used to close rather large defects in the muscular wall of the esophagus after removal of leiomyomas. In Patient B, operated upon in 1954, a tumor of the esophagus was removed (Figs. 1 and 2) and a long, narrow graft (3 by 12 cm.) of the diaphragm was used to close the defect. A postoperative roentgenogram (Fig. 3) showed a good lumen of the esophagus and full involvement of the graft in peristalsis. At the present time, 4½ years

after surgery, the patient is in good condition, and repeated x-ray studies have failed to show any noticeable traces of the operation. In all 10 cases the immediate and long-term results of surgery have been good (from 1 to 6 years).

Similarly, good results have been obtained with grafts following the removal of an esophageal diverticulum in the lower esophagus. Besides bulging

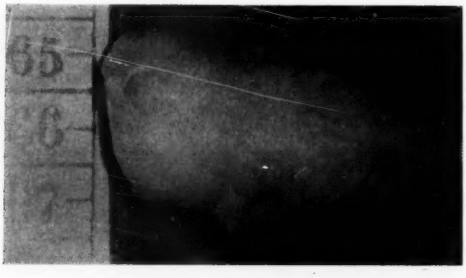

Fig. 2.—Removed leiomyoma.

Fig. 3.—Patient B. Roentgenogram of the esophagus made 1 month after surgery.

of the esophageal wall, we observed considerable thinning of the muscular layers around the diverticulum, leading to recurrence in 2 of our patients. Strengthening of the muscular tissue, both in the suture area and in its circumference, is very important. Six patients with diverticulum of the esophagus have been operated upon using diaphragmatic grafts.

Diaphragmatic grafts have also been found very useful in operations for cardiospasm. Esophagofundostomy has been followed by poor long-term results, such as esophagitis, ulcer, or hemorrhage, and even resection of the cardia cannot relieve the patient from the serious and dangerous regurgitation which is responsible for the complications just mentioned. Our method consists in the section or resection of the scarred muscular areas of the lower esophagus and the upper stomach to be followed by closure of the defect with a triangle-shaped diaphragmatic graft.

The edges of the graft are sutured to the wall of the esophagus, followed by suturing the graft at its base to the medial and lateral surfaces of the diaphragm. Thus the graft is spread so that it prevents the processes of scarring and fibrosis in the area of operation. One can use other methods for closing the defect following section of the scarred muscular wall of the esophagus by placing the diaphragmatic graft transversely. The immediate effect of surgery is excellent, and good results have been observed in 45 patients followed for 2½ years or less (Figs. 4, 5, 6).

A diaphragmatic graft was also used on a patient who suffered from an esophageal fistula and empyema which developed after ulcerative esophagitis of undetermined etiology. The fistula was closed with a diaphragmatic graft affixed to a pedicle and the empyema was eliminated by lower thoracoplastic surgery.

In 2 cases diaphragmatic grafts were used to correct a hernia through the esophageal opening and to control the concomitant phenomenon of gastroesophageal regurgitation. In these cases the graft was wrapped around the area of the absent cardiac sphincter, thus serving as a muscular casing which substituted for the cardia. This casing functioned very well in one instance, whereas in the second case we noted certain regurgitation phenomena. Another indication for the diaphragmatic plastic operation is the closing of the lung wound and the bronchial stump after pneumonectomy. The bronchial stump and pulmonary wound were closed with a free diaphragmatic graft in 16 cases. No bronchial fistulas occurred.

Wounds of the liver were closed successfully with diaphragmatic grafts in 2 patients. Diaphragmatic grafts were used also in our experimental work to close wounds of the heart and aorta.

After checking this method in our experimental work, we used it successfully in surgery of aneurysm of the heart in 8 cases. It is specially indicated in aneurysms of the left ventricle. With the patient lying on his right side, a thoracotomy is performed through the sixth intercostal space. The pericardium is cut rather far away from the aneurysm. The aneurysm is usually easily seen and can be felt by palpation. Auscultation may disclose murmurs which can be recorded on the Mingograf. The aneurysm is a circular bulging of the cardiac wall, the surface of which is scarred and white colored. The adjacent portions of the heart are also scarred. The vessels of the epicardium cannot be detected in this area. Palpation will disclose thinning of the myocardium at the apex of the aneurysm. It is rather difficult to detect thrombi

Fig. 4.

Fig. 5.

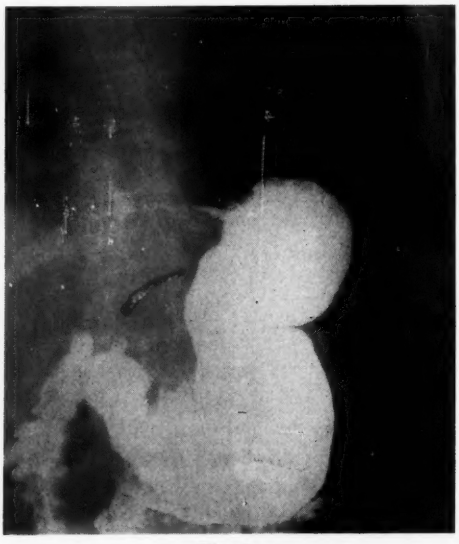

Fig. 6.

Fig. 4.—Patient T. Cardiospasm. Roentgenogram which shows esophageal dilatation prior to surgery.

Fig. 5.—Patient T. Roentgenogram of esophagus made after surgery.

Fig. 6.—Patient T. Roentgenogram made after surgery with the patient in the Trendelenburg position to show absence of regurgitation.

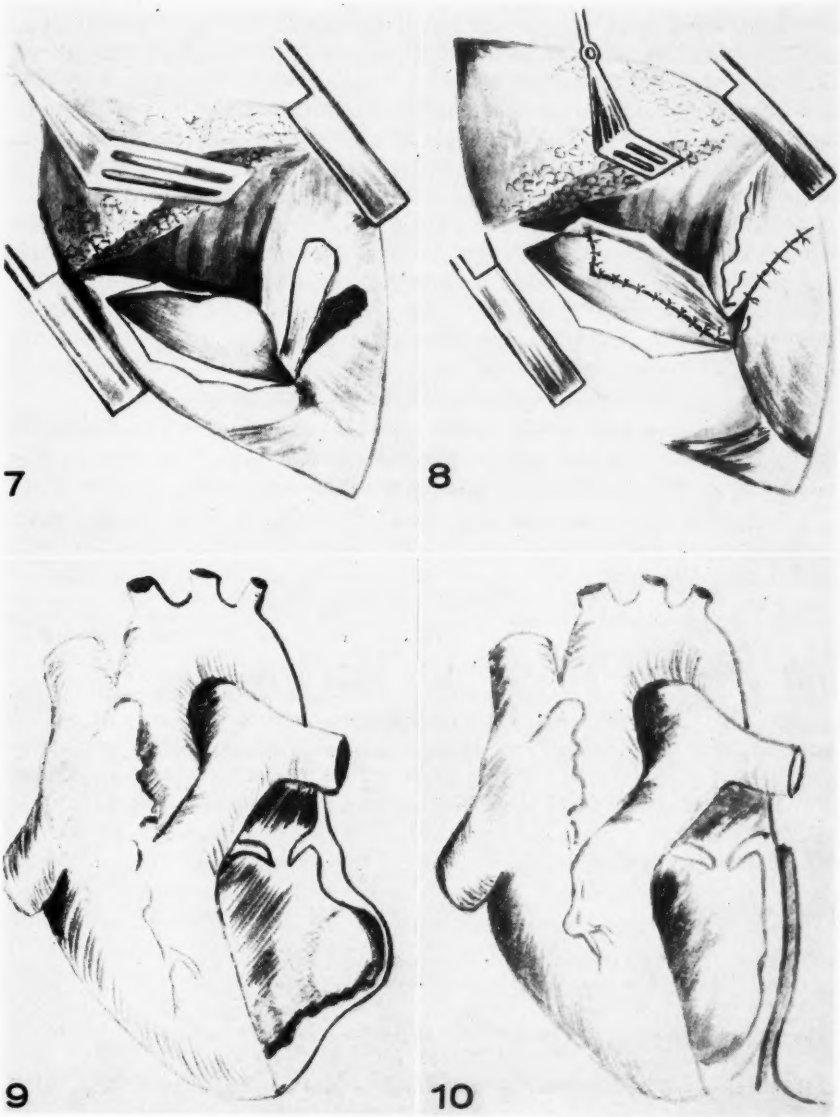

Fig. 7.—Cutting out of diaphragmatic grafts.
Fig. 8.—Suturing of diaphragmatic graft to cardiac muscle in the area of the aneurysm.
Fig. 9.—Aneurysm of the heart.
Fig. 10.—Flattening of the aneurysmal base after suturing of diaphragmatic graft.

in the lumen of the aneurysmal sac but we must always bear in mind their possibility and be very careful when operating on the aneurysmal sac itself.

After auscultation and palpation we determine the borders of the aneurysm and the state of the coronary vessels in the vicinity of the aneurysm. A pedicled graft is cut from the muscular part of the diaphragm, 12 by 6 cm. in size. The base of the graft must stick tightly to the apex of the heart. After thorough hemostasis of the diaphragm and edges of the graft, it is turned to the apex, and the sutures to be applied to the heart are outlined. The surface of the graft and the epicardium in the area of the aneurysm are carefully and sparingly scarified with a sharp spoon. The graft is sutured to the muscle of the left ventricle, covering the aneurysm. In the transverse direction the sutures are usually applied under tension which makes the base of the aneurysm flat and practically corrects it altogether (Figs. 7, 8, 9, 10). After this manipulation, auscultation usually demonstrates the disappearance of murmurs. In 1 case prior to the plastic operation the aneurysm was corrected by suturing it with tantalum staples by means of a special suturing apparatus (UKL).

In our 8 cases of aneurysm of the heart, the application of diaphragmatic grafts has been quite successful. The patients tolerated the operation well and were in good condition from 3 to 10 months after surgery.

In Table I are summarized our data on the 105 cases of diaphragmatic plastic operations.

TABLE I. THE USE OF DIAPHRAGMATIC GRAFTS IN THORACIC SURGERY

TYPE OF DISEASE	METHODS USED IN APPLICATION OF DIAPHRAGMATIC GRAFTS	NO. OF CASES
Cancer of the cardia and esophagus	The covering of the gastrointestinal or gastroesophageal anastomoses with diaphragmatic grafts	15
Cardiospasm	Plastic operation on the cardia by using diaphragmatic graft	45*
Benign tumors and cysts of the esophagus	Closing of esophageal wall defect with diaphragmatic graft	10
Epiphrenal diverticulum of esophagus	Closing of esophageal wall defect with diaphragmatic graft	6
Esophageal fistula	Closing of esophageal wall defect with diaphragmatic graft	1
Gastroesophageal regurgitation in cases of hernia of esophageal orifice	Creation of cardiac sphincter from diaphragmatic graft	2
Lung: cancer and suppurative disease	Closing of lung wound or bronchial stump with diaphragmatic graft	16
Liver: wounds and trauma during surgery	Closing of wounds with diaphragmatic graft	2
Heart: aneurysm of the heart, after infarction	Plastic surgery of aneurysm with application of diaphragmatic graft	8†
Total		105

*June 23, 1960.—At this time, an additional 15 operations of this type have been performed, with no deaths.

†June 23, 1960.—At this time, we have performed an additional 15 operations of this type. Twelve of them were with total removal of the aneurysm, suture, and diaphragmoplasty. Two patients have died in 23 operations.

Further elaboration of this method in our experimental work demonstrated possibilities of its wider application. For example, experiments conducted by T. A. Suvorova showed that the stump of the bronchus can be closed with a diaphragmatic graft affixed to a pedicle without applying any sutures to the bronchus. She also elaborated on the method of closing fresh wounds of the esophagus, which is of obvious practical significance. J. A. Natsvlishvili and I. Z. Kozlov studied the technique of closing defects of the myocardium with diaphragmatic grafts. These experiments, together with the experimental work conducted at some clinics abroad, testify in favor of using such diaphragmatic plastic operations in revascularization of the myocardium, in aneurysms of the heart and in the closing of wounds after removal of tumors of the heart. It is now clear that this type of plastic surgery should be used on a broader scale in thoracic surgery.

SUMMARY

1. Animal experiments demonstrated extremely valuable qualities of the diaphragm for use in plastic surgery. Grafts of the diaphragm affixed to a pedicle and cut out with due consideration for their innervation and blood supply grow very well into the pericardium, cardiac muscle, aorta, lung, esophagus, and liver.

2. The clinical application of this method, begun in 1948, yielded good results in operations on the esophagus for tumors, cysts, and diverticula. Here we noted the possibility of substituting the vascular wall of the esophagus with the diaphragmatic muscle.

3. Good results were obtained in the application of diaphragmatic grafts affixed to a pedicle for closing the bronchial stump.

4. In aneurysm of the heart this method enabled us to improve the vascularization of the myocardium and to strengthen the scarred wall of the heart with diaphragmatic muscle.

5. This method also is used in the following situations: closing defects of the wall or strengthening the line of anastomosis in operations on the esophagus; plastic operations on the cardia for cardiospasm; creating artificial cardiac sphincter in gastroesophageal regurgitation; and for closure of wounds of the lung, liver, heart, and aorta incurred during operations on these organs.

6. Observations and results in 105 cases in which diaphragmatic grafts were employed are presented.

CLINICAL AND PHYSIOLOGIC CONSIDERATIONS OF LEFT HEART BYPASS DURING CARDIAC ARREST

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PROLONGED, unsuccessful, intermittent cardiac compression ("cardiac massage") is associated with an increasingly hypoxic and unresponsive myocardium. Manual systole is unable to provide a fully adequate coronary blood flow. In contrast, markedly hypoxic hearts may be speedily revived after elective arrest by means of a pump-oxygenator system. The mechanical system provides a nearly normal systemic pressure and coronary blood flow, and spares the myocardium the trauma of manual systole. In an effort to improve the salvage obtained by conventional methods of emergency, cardiac resuscitation, an investigation of the use of a mechanical pump to bypass the left heart was carried out. This technique provides adequate coronary blood flow, adequate systemic circulatory support, and is suitable for emergency application.

The technique of left heart bypass consists of mechanically pumping oxygenated blood from the left atrium to a systemic artery (Fig. 1). This system has been used to shunt blood around isolated segments of the thoracic aorta[3, 7] and to support the left ventricle during its recovery from elective arrest during open-heart surgery. For these applications a functioning right ventricle circulates blood through the ventilated lungs to the left atrium.

The work of Starr, Jeffers, and Meade[24] and, later, others[22] suggested that the right heart might not be required for perfusion of the pulmonary circuit. They found that the systemic circulation was maintained in the absence of a beating right ventricle with only a moderate elevation of venous pressure.

Stimulated by this work, Jamison and his co-workers[15] instituted a left heart bypass circuit in 10 dogs and then caused the heart to fibrillate. Using vasopressors to increase both peripheral vascular resistance and systemic venous pressure, they were able to perfuse animals for up to 45 minutes at normal arterial pressures with survival. They did not report flow rates, but commented that, at high flows, the left atrial catheter tended to collapse.

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Helmsworth and associates[14] reinvestigated Jamison's work. They drained the left atrium by gravity into a reservoir and omitted the vasopressors. They found that an elevated venous pressure was necessary in this extracorporeal circuit to assure adequate pulmonary blood flow. The venous pressure was raised by over-transfusion. An increase of 50 to 75 per cent of the dog's estimated blood volume was required to attain flows of 100 c.c. per kilogram per minute. However, they abandoned the procedure because of technical difficulties, probable

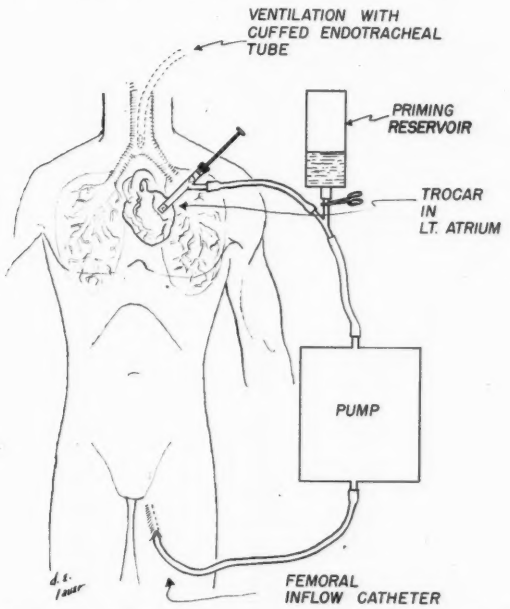


Fig. 1.—Diagram of the extracorporeal system. The reservoir is usually clamped except when blood or plasma expander is added.

inapplicability in patients with increased pulmonary vascular resistance, and the occurrence of serious postoperative hypotension in their dogs. Drew[9] also mentioned that he abandoned the use of the left heart bypass alone because of difficulties with the left atrial return.

We became interested in the clinical applicability of the left heart bypass technique for cases of unexpected cardiac arrest in which manual systole had been unsuccessful. In conjunction with a continuing clinical series, experiments were carried out in 36 dogs to study modifications of the technique and the relevant physiology.

EXPERIMENTAL DESIGN

Healthy mongrel dogs were anesthetized with intravenous sodium Pentothal and intubated with a cuffed endotracheal tube. The chest was opened through the left fourth interspace. Animals were given heparin intravenously, 1 mg.

per kilogram. Aortic and vena caval pressures were monitored in all dogs, and in 14 animals pulmonary arterial and venous pressures were also recorded on a multichannel, direct writing oscillograph. A No. 12 or 14 Fr. inflow catheter was placed in the femoral artery. The left atrium was catheterized with a specially designed trocar (Fig. 2) which permitted rapid cannulation without an initial purse-string suture. This trocar was used in 25 dogs without complication. Lung aeration was maintained by an Emerson respirator or by manual bag breathing.

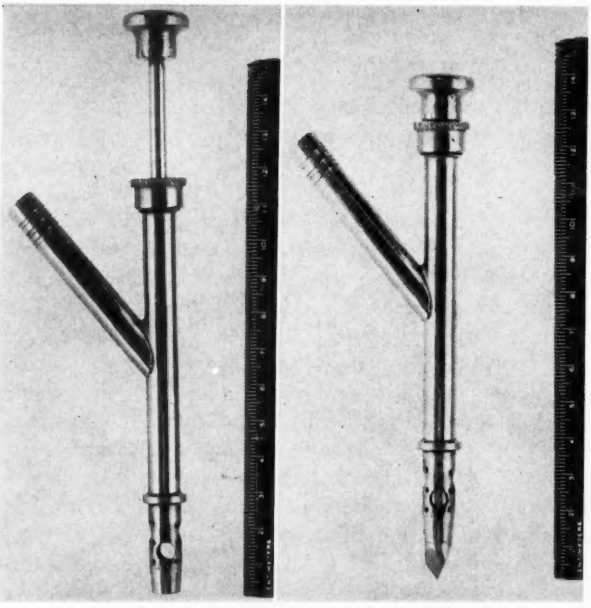


Fig. 2.—Photograph of the atrial trocar. The atrium is lifted by three Allyce clamps and the trocar thrust through the wall. A ligature secures the trocar in place. The obturator is then withdrawn and, when the side arm is filled with blood, it is connected to the venous line. Neoprene washers insure an air-tight system.

The perfusion circuit is illustrated in Fig. 1. Tygon tubing was led from the atrial trocar to a Sigmamotor pump and thence to the femoral arterial inflow catheter. Flows were determined by calibration of the pump and checked by direct measurement into a graduate from a Y in the arterial line. Blood or plasma expander was added intermittently from a reservoir connected to the venous line. During perfusion the heart was stopped or caused to fibrillate by a variety of means while the coronaries were perfused by the extracorporeal system. Electrical defibrillation after 30, 60, or 90 minutes of perfusion was attempted in some dogs. Twenty-eight dogs were perfused at least 60 minutes. In 7 dogs, blood chemistries were drawn (Table I) and lung and liver biopsies taken after 30 and 60 minutes of bypass. Chemical determinations were made according to standard clinical techniques. Long-term survival was attempted in 3 dogs. In 6 animals the effects of norepinephrine or Regitine were studied.

RESULTS

Immediately after initiating bypass, addition of blood or plasma expander was required to maintain adequate flow (60 c.c./Kg./min.) into the left atrium. The addition of 20 to 40 per cent of the animal's blood volume, which was assumed to equal 10 per cent of the body weight, elevated the systemic venous pressure to 18 to 25 mm. Hg (Fig. 3). The gradient between the systemic venous pressure and the pulmonary arterial pressure never exceeded 3 mm. Hg. Initially a near normal, though nonpulsatile, pulmonary arterial pressure was required to allow adequate flow through the lungs. Later when the preparation became stabilized and pulmonary resistance decreased, less pulmonary arterial pressure was required to maintain equivalent flow.

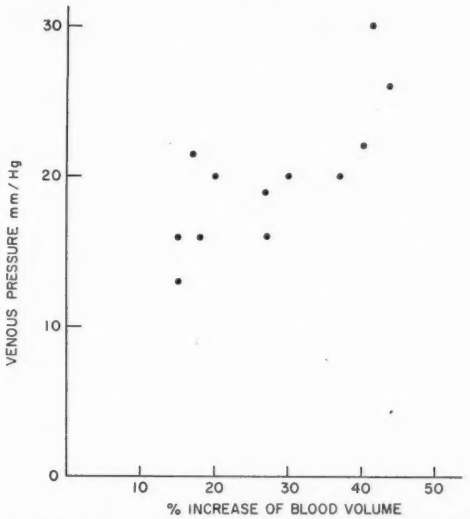


Fig. 3.—Venous pressure rises in proportion to the degree of hypervolemia. However, there is considerable biologic variation in the pressure achieved for a given blood volume increment.

The elevated systemic venous pressure resulted in an apparent loss of intravascular fluid volume at the rate of 1.0 to 1.5 c.c. per kilogram per minute. This rate was determined by the amount of blood or dextran that was required per minute to maintain the elevated venous pressure at a constant level during the perfusion. The elevated venous back pressure did not produce any grossly apparent effect on the peripheral resistance. However, if a large opening was made in the pericardium, the elevated venous pressure caused dilatation of the right heart. Right heart dilatation did not occur with a limited incision which preserved the pericardial envelope.

Since the pressure differential across the right heart never exceeded 3mm. Hg, the major impedance to flow was the pulmonary vascular resistance. This functionally cardioplegic preparation afforded a unique opportunity to study the factors affecting pulmonary vascular resistance. Fourteen experiments were carried out for this purpose.

If the pulmonary venous pressure (left atrial pressure) became less than 5 to 7 mm. Hg, the catheter from the left atrium collapsed and pulmonary venous return virtually stopped. This observation confirms the "critical closing pressure" predicted[6] and observed[4] by others. However, if the animal was made hypervolemic by transfusions of 30 to 40 per cent of its estimated blood volume, this critical closing phenomenon disappeared. High flow rates continued in spite of pulmonary venous pressures down to 1 or 2 mm. Hg (Fig. 4). At low flow rates (35 c.c./Kg./min.) critical closing was always seen at left atrial pressures below 3 mm. Hg.

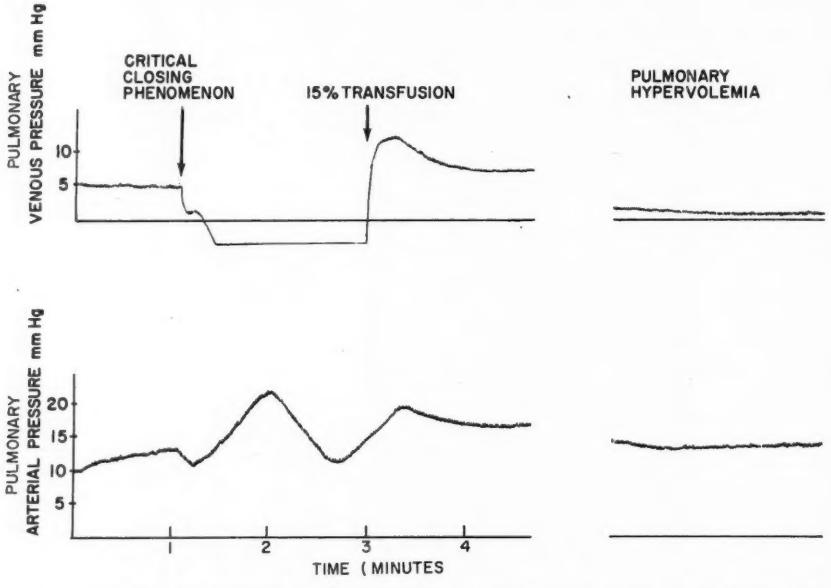


Fig. 4.—These are tracings of pulmonary arterial and venous pressures at a flow rate of 52 c.c./Kg./min. In the isovolemic or slightly hypervolemic animal, left auricular tubing collapsed and flow ceased when the pulmonary venous pressure reached 5 mm. Hg. Pulmonary venous pressure was restored with a large transfusion. Later in the same animal with a greatly increased blood volume and pulmonary hypervolemia, flow continued at pulmonary venous pressures of 2 mm. Hg.

In this preparation pulmonary vascular resistance varied with flow rate (Fig. 5). At low flows, pulmonary vascular resistance was great, while at high flow rates the resistance was low. Furthermore, at a constant flow rate, the pulmonary vascular resistance gradually decreased over a period of time (Fig. 6). Since the flow rate was not changed, the decrease in pulmonary vascular resistance was accompanied by a proportionate decrease in the pressure differential across the lungs (ΔP). Fig. 6 shows that the decrease in ΔP is related to either a decrease in pulmonary arterial pressure or an elevation of pulmonary venous pressure. Both of these mechanisms were observed in the dog. At least a 40 per cent decrease in pulmonary vascular resistance was seen in these animals within 15 minutes of beginning a steady flow rate.

The absolute level of pulmonary vascular resistance varied a great deal from animal to animal. However, all animals showed the relative pressure-flow relationships just noted. These experiments do not test Haddy's[12] and Borst's[4] findings that, at pressures above critical closing, pulmonary vascular resistance decreases when left atrial pressure is increased. When flow rates were held relatively constant, our data are perhaps suggestive that pulmonary vascular resistance may decrease at higher pulmonary venous pressures (Fig. 7). However, the marked scatter of the data precludes a definite conclusion.

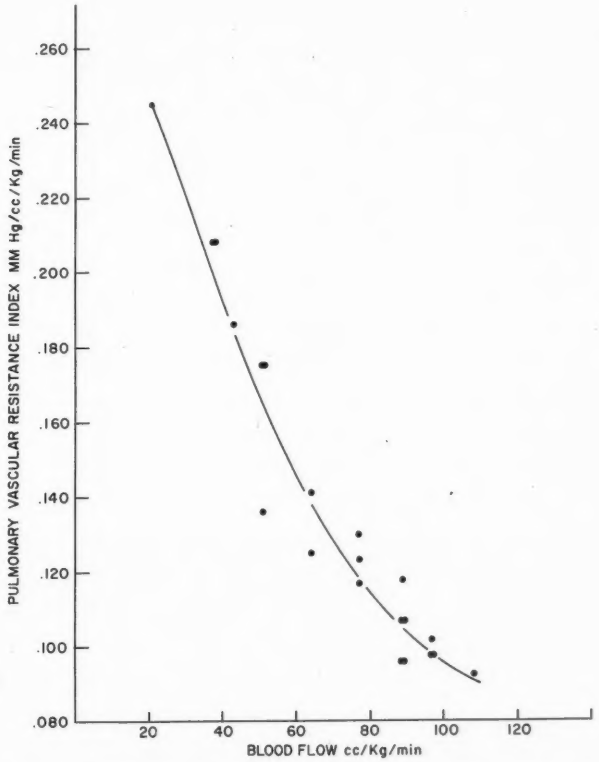


Fig. 5.—Pulmonary vascular resistance index is calculated as the difference between pulmonary arterial and venous pressures divided by the pulmonary flow rate per unit body weight. The above data from a single experiment show a consistent decrease in pulmonary vascular resistance with increasing flow rates.

In 6 dogs, norepinephrine or Regitine (phenolamine) added systemically or into the pulmonary artery in physiologic doses (less than 1 mcg./Kg./min. of norepinephrine) did not alter pulmonary vascular resistance. Doses of norepinephrine above 1 mcg. per kilogram per minute simulated a transfusion by constricting the peripheral vascular bed and thus increased both systemic arterial and venous pressures.

TABLE I. CHEMICAL DETERMINATIONS IN 7 DOGS*

	CONTROL	½ HOUR	1 HOUR
O_2 Saturation	—	95.5 (95 to 100)	92 (90 to 95)
pH	7.55	7.26 (7.15 to 7.34)	7.19 (7.02 to 7.38)
CO_2 (mEq./L.)	15	12.8 (8 to 15)	10.8 (2 to 14)
NA^+ (mEq./L.)	139	140 (138 to 140)	142 (140 to 144)
K^+ (mEq./L.)	4.0	3.7 (2.8 to 4.2)	3.3 (2.5 to 4.3)
Cl^- (mEq./L.)	106	119 (112 to 124)	119 (107 to 137)
Pyruvate (mg./100 c.c.)	3.0	4.0 (2.8 to 5.0)	4.0 (1.8 to 5.6)

*The flow rate varied between 40 and 85 c.c. per kilogram per minute.

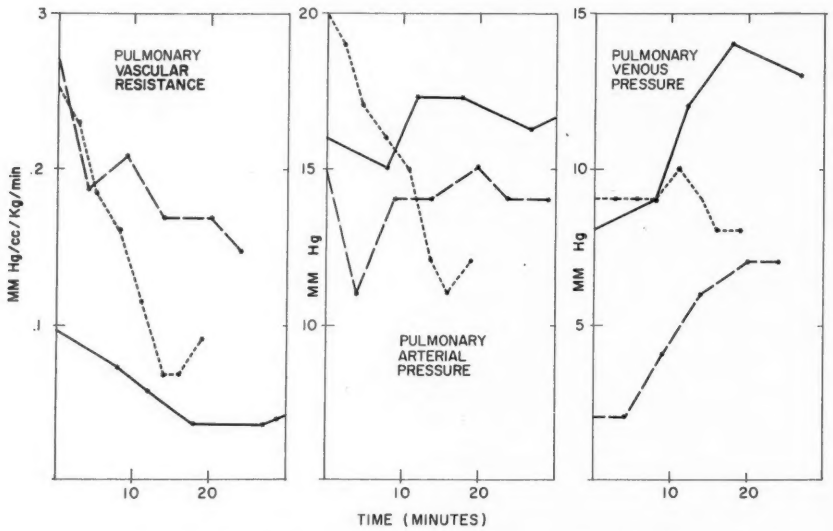


Fig. 6.—These graphs show the pulmonary vascular measurements plotted against time in three different animals perfused at a constant flow rate. In all animals the pulmonary resistance decreased at least 40 per cent in the first 15 minutes of perfusion. The pulmonary arterial pressures *(middle graph)* were held nearly constant in 2 dogs by replacement transfusion. Note that the pulmonary venous pressures in these 2 dogs increased, resulting in a decrease in pulmonary resistance. The other dog was not transfused during this period, and the pulmonary venous pressure remained stable while the pulmonary arterial pressure decreased causing a similar decrease in pulmonary resistance.

Chemical studies were carried out on 7 dogs before perfusion, and after 30 and 60 minutes of bypass (Table I). At flows above 60 c.c. kilogram per minute, although oxygen saturation tended to decline slightly during the hour of bypass, saturation was always maintained above 90 per cent. The decline in oxygen saturation coincided with a metabolic acidosis which developed during the perfusion. Flow rates above 60 c.c. per kilogram per minute tended to minimize the acidosis, but could not prevent it. Furthermore, the acidosis occurred despite the maintenance of a low pCO_2 by mechanical ventilation with 100 per cent oxygen in line with a CO_2 absorber. Low CO_2 values were accompanied by elevation of blood chloride values. Pyruvate levels increased slightly, but this increase did not account for the acidosis nor was it consistent in all dogs. Sodium and potassium values were not significantly altered by the perfusion.

Lung biopsies after 30 and 60 minutes were normal (Fig. 8). Grossly the lungs were neither edematous nor difficult to ventilate. Liver biopsies showed moderate congestion with extravasation of red cells into the sinusoidal spaces (Fig. 8). Grossly the liver was engorged. In 4 of 19 dogs, 200 to 400 c.c. of slightly cloudy, but not sanguine, ascitic fluid was recovered. No gross intestinal hemorrhage or pathologic changes were noticed.

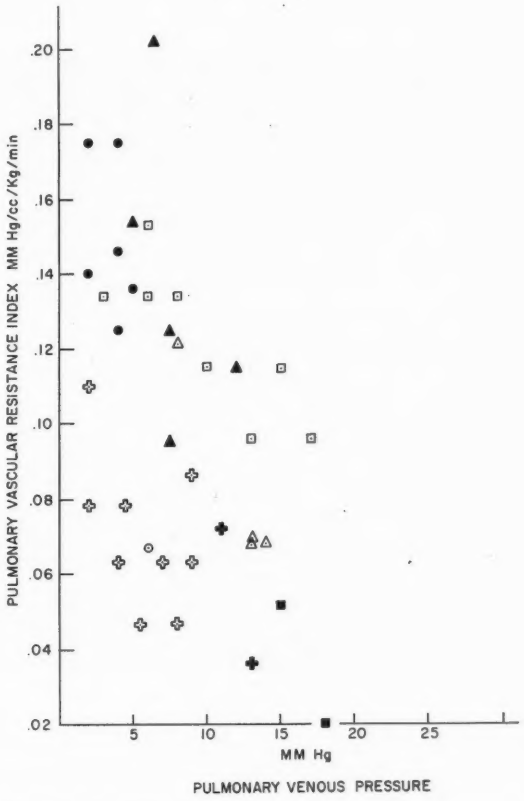
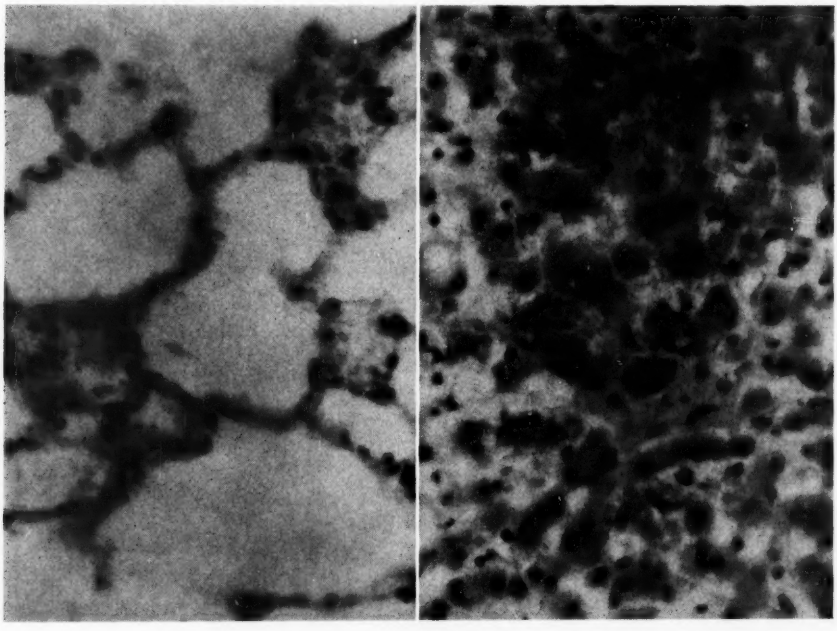


Fig. 7.—Pulmonary vascular resistance is plotted against pulmonary venous pressure in 8 dogs whose flow rates were held between 50 and 65 c.c./Kg./min. A different symbol is used for each dog. Our data do not permit a definite conclusion as to the relationship of these two variables.

In five acute experiments in which defibrillation was attempted, the hearts readily took over and adequately maintained the circulation. To test further the ability of the preparations to survive, left heart bypass was performed on 3 dogs under aseptic conditions, using cold to produce ventricular fibrillation. In 1 dog, bypassed for 45 minutes during ventricular fibrillation, the pericardial sac was opened widely allowing the right heart to dilate. This dog lived 8 hours but died of congestive heart failure. In 2 other dogs, the heart was confined by the pericardium. These animals were bypassed for one-half

hour each, recovered promptly after defibrillation, and were long-term survivors without detectable organ damage. Perfusion at 60 c.c. per kilogram per minute maintained their blood pressure above 100 mm. Hg. After restarting the heart, the venous pressure was lowered to pre-perfusion levels by withdrawal of venous blood. The amount recovered was approximately equal to the amount transfused, minus the extravascular loss calculated at 1.0 to 1.5 c.c. per kilogram per minute. Electrocardiograms did not show any evidence of injury in these dogs.



A. *B.*

Fig. 8.—Photomicrographs of lung *(A)* and liver *(B)* after 1 hour of left heart bypass perfusion during cardiac arrest. There is no edema or extravasation of red cells within the lungs, although vessels are filled. The liver slide, however, shows moderate congestion with extravasation of red cells into the sinusoidal spaces.

DISCUSSION

These experiments define the conditions under which left heart bypass alone will sustain the circulation during cardiac arrest. This confirms the work of Jamison[15] and Helmsworth.[14] However, the unphysiologic features of the procedure limit it sharply. Multiple blood or plasma substitute transfusions are required to elevate the systemic venous pressure. Peripheral vasoconstrictors decrease the number of transfusions required and raise the arterial pressure, but may decrease organ blood flow to undesirably low levels.

The elevated venous pressure results inevitably in acute hepatic congestion. Brewin and his colleagues[5] found that hepatic congestion for even 10 minutes

resulted in a depressed, post-congestion, galactose tolerance test and in an increased metabolic acidosis. However, as demonstrated by patients with chronic right heart failure, elevated systemic venous pressure and hepatic congestion can be tolerated without severe metabolic difficulty.

Only a portion of the decline in arterial oxygen saturation can be explained by the shift of the oxygen saturation curve to the right with acidosis.[21] Pulmonary hypervolemia and changes in pulmonary blood flow during perfusion may account for the rest.

The metabolic acidosis occurred in spite of hyperventilation and increased removal of CO_2. Although pyruvate levels did not consistently increase, hepatic congestion and tissue hypoxia resulting from marginally adequate flow or from redistribution of the circulation offer sufficient explanation. While the acidosis might be minimized by the addition of base to the perfusate,[19] it is progressive with time and, therefore, it seems advisable to limit perfusion time to 1 hour.

Although left heart bypass in the presence of an arrested heart presents hemodynamic abnormalities which probably cannot be tolerated for much over 1 hour, this technique offers several important advantages in cases of unexpected cardiac arrest. The simplicity of the extracorporeal circuit and low priming volume permit early initiation of perfusion. A kit of instruments, suitable catheters, the atrial trocar, and mounted Tygon tubing can be sterilized and stored for immediate use. A pump, anesthesia machine, and ordinary bank blood complete the necessary equipment. While intermittent cardiac compression is carried out, the left atrial and femoral arterial catheters are quickly inserted, and the patient is rapidly transfused to increase the systemic venous pressure. An experienced team can initiate perfusion within 15 or 20 minutes of arrest.

Left heart bypass perfusion does not replace intermittent compression in the cardiac resuscitative program. Rather, it is a supplementary procedure applicable to those hearts which do not respond to massage, conventional drugs, or defibrillation. The time needed for the initiation of the bypass allows an adequate trial of these less elaborate procedures. However, prompt installation of left heart bypass perfusion insures an adequate coronary and systemic blood flow and spares the myocardium additional trauma from prolonged, intermittent compression.[1]

Left heart bypass has been applied clinically in 6 cases. One patient with a ruptured aortic aneurysm was perfused for 12 minutes. This man subsequently lived 35 days. Three other severely hypoxic, arrested hearts were perfused up to 1 hour. All of these hearts were successfully defibrillated and regained adequate contractions, but failed again in the postoperative period. Two patients with massive myocardial infarctions complicated by ventricular fibrillation underwent emergency coronary thrombendarterectomy. Left heart bypass sustained the systemic circulation for over 2 hours. Although the coronary vessels were cleared and defibrillation accomplished, these hearts were unable to maintain the circulation at the conclusion of the procedure.

These experiments with left heart bypass add data concerning the regulation of pulmonary vascular resistance. Within the limits of the experiments, no neural or humoral control of pulmonary vascular resistance was observed. The decline in pulmonary resistance seen with increasing pulmonary blood flow confirms Williams' observations.[25] The critical closing phenomenon predicted by Burton[6] and observed by Borst[4] and others in the isovolemic animal was confirmed.

During perfusion at a constant flow rate, pulmonary vascular resistance decreased. Further, when the animal was hypervolemic, the critical closing phenomenon disappeared. The work of Sjostrand[23] and Gauer, Henry, and Sieker[11] has bearing on the absence of critical closing phenomena in the hypervolemic animal. They showed that the thoracic viscera, and especially the lungs, constitute a great intravascular reservoir which may sequester large volumes of blood with very little elevation of intravascular pressures. This reservoir is not influenced by mild positive pressure breathing.[20] Pulmonary hypervolemia causes dilatation of the pulmonary vasculature and particularly of the thin-walled pulmonary veins. In the isovolemic animal these veins are undistended and collapse when their intraluminal pressure decreases below a certain critical pressure. Overtransfusion and the resulting pulmonary engorgement may distend these veins and thus limit the critical closing phenomenon. Data from Kantrowitz and Kantrowitz[17] further support this explanation. They found that satisfactory pulmonary blood flows could be obtained in cats at negative pressures up to 30 mm. Hg with steel cannulae in the major pulmonary veins. Thus, the requirement for continuous addition of blood or plasma expander during perfusion may be accounted for by storage in the pulmonary and systemic vascular reservoirs as well as accumulation of edema and ascites.

SUMMARY

The systemic circulation can be maintained during cardiac arrest by pumping blood from the left atrium to a systemic artery. This left heart bypass technique has been investigated in dogs and used in human beings. The systemic venous pressure is raised by transfusions to permit adequate blood flow through the right heart and ventilated lungs. Pulmonary vascular resistance is decreased by hypervolemia, elevation of pulmonary venous pressure, or increased rate of blood flow. At a constant flow rate, pulmonary vascular resistance decreases with time. Hepatic congestion and a moderate metabolic acidosis occur after 1 hour of perfusion.

The simplicity of this extracorporeal system recommends its use as a supplementary procedure in cases of unexpected cardiac arrest not responding to conventional measures. This technique has been applied with promising results in 6 clinical cases.

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AN IMPROVED ELECTROMAGNETIC FLOWMETER FOR EXTRACORPOREAL CIRCULATION

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ACCURATE FLOWMETERS for use with extracorporeal circulation units have not previously been available. Blood flow, when measured, has been recorded in some indirect fashion. During the past 18 months we have been working with an instrument which seems to fulfill the accepted requirements for a flowmeter for use with artificial heart-lung devices and other extracorporeal circulation units. Laboratory studies and clinical applications form the basis for these opinions. This report describes the flowmeter and defines its use.

DESCRIPTION AND PRINCIPLE OF THE MAGNETIC FLOWMETER†

The complete unit consists of a transmitter which senses blood flow rate and an electronic receiving instrument which indicates, records, and integrates the measurement during bypass.

Faraday's principle of electromagnetic induction, on which the flowmeter is based, states that when a conductor moves through a magnetic field a voltage is generated in direct proportion to the velocity of the conductor.

Expressed mathematically, $E = C\ hdv$, where E is the generated voltage, C is a dimensional constant, h is the magnetic field strength, d is the diameter of the conductor, and v is the velocity of the conductor.

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†Foxboro Electromagnetic Flowmeter, The Foxboro Company, Foxboro, Mass.

Transmitter.—The blood flow transmitter (Figs. 1 and 2) consists of a straight Teflon tube, ⅜ inch in internal diameter, sheathed by a stainless steel jacket, which conducts blood through a magnetic field. The Teflon tube is provided with atraumatic end connectors for ½ inch and ⅜ inch plastic tubing.

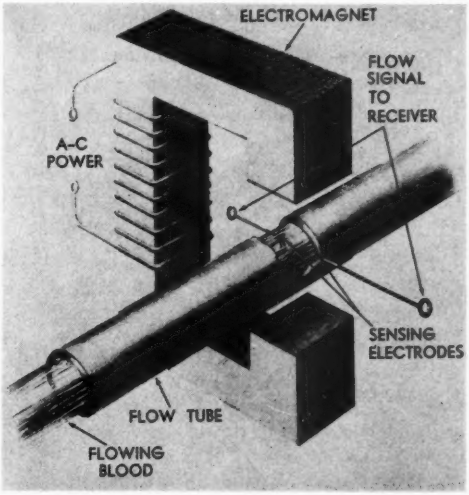

Fig. 1.—Schematic drawing of the transmitter.

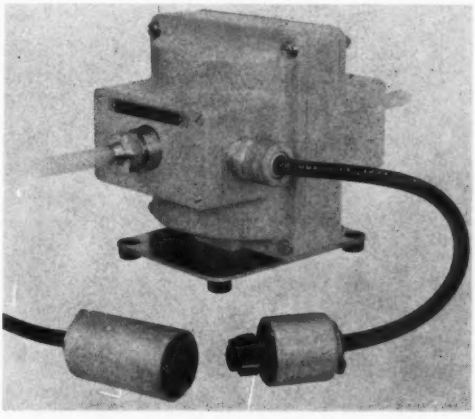

Fig. 2.—Transmitter assembled, ready for clinical use.

A uniform magnetic field is produced by an electromagnet positioned so that the lines of force pass perpendicularly through the ⅜ inch flow tube. As the blood flows, it intersects the lines of force, generating a voltage which is linear and proportional to the average velocity of the flowing blood. This voltage is sensed by two stainless steel "point" electrodes which are mounted flush with the interior of the tube in a plane perpendicular to the magnetic field and the

axis of the tube. The measurement signal is conducted to a Dynalog Receiver through a two-wire shielded cable. Since average velocity is proportional to volume rate of flow, the instrument is calibrated to read directly in liters per minute.

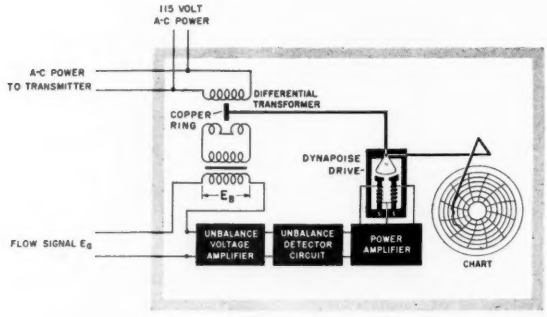

Fig. 3.—Schematic drawing of the receiver.

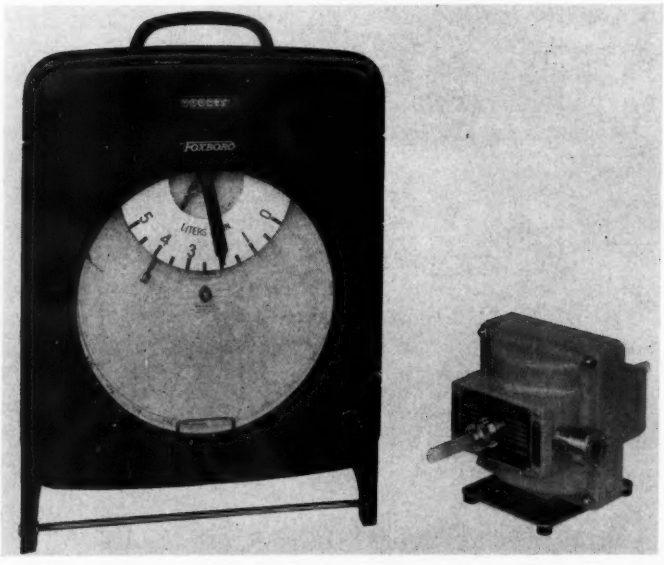

Fig. 4.—Receiver. Note large dial for indication of flow rate. Transmitter is also shown.

Density, turbulence, viscosity and suspended materials, as well as temperature, pressure changes and conductivity, do not affect the generated voltage. Deposits on the inner surface of the tube do not modify the performance. The pressure drop across the flowmeter is the same as is encountered through a smooth bore plastic tube of the same size (⅜ inch internal diameter) and length (25 inches).

Receiver.—At the Dynalog Receiver (Figs. 3 and 4) the measurement voltage (E_G) from the transmitter is connected in series opposition to a balancing voltage (E_B) produced by a differential transformer. Transformer output varies with the position of a copper ring linked to the drive shaft of a Dynapoise magnetic drive unit. Also linked to the shaft is the receiver pen movement.

When the receiver pen is at rest on the correct reading, voltages E_G and E_B are equal and opposite; hence the difference between them is zero. With an increase or decrease in blood flow rate, E_G changes, and the resulting difference between the opposed voltages becomes a net value directly proportional to the change in flow rate. This net value or voltage unbalance goes to the voltage amplifier where it is amplified and then passed through a detector circuit to determine the direction of measurement change.

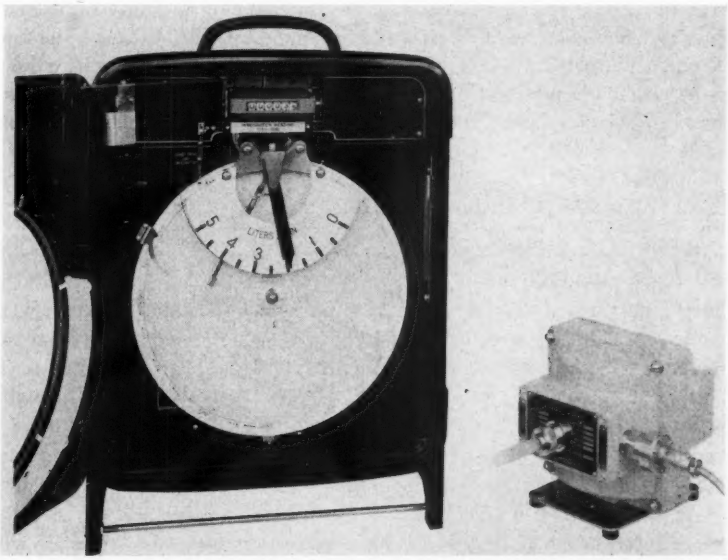

Fig. 5.—Circular recording chart on receiver. Direct writing pen makes continuous recording of flows.

The output of the detector circuit, after power amplification, energizes a solenoid motor in the Dynapoise drive unit causing the pen to travel upscale or downscale. The Dynapoise drive also repositions the copper ring in the differential transformer so that the balancing voltage once again equals the measurement voltage. Since this balancing action is practically instantaneous, any changes in flow rate are indicated and recorded as they occur.

Both the differential transformer and the flowmeter are supplied from the same power source (115 volt alternating current). This allows line voltage changes of plus or minus 10 per cent with no effect upon the instrument's calibration since both generated and balancing voltage change in unison. The two

cables connecting the flowmeter to the recorder can be about 100 feet long enabling the recorder to be placed at that distance away from the flowmeter. The direct recording pen inscribes on a circular chart which makes one rotation per hour (Fig. 5). There is an indicator mounted in the upper part of the recorder that enables direct reading at considerable distances (see Fig. 4), allowing the surgical team to have a constant evaluation of the flows. The recorder also is equipped with a "totalizer" which gives a cumulative sum of the total volume flow during the bypass. To avoid pulse fluctuation produced by the pumps used in extracorporeal circulation, a special recording pen dampener has been incorporated, delaying the full scale excursion of the pen to 20 seconds.

The total range of the recorder is 5.6 liters (1.5 gallons). The calibration accuracy is plus or minus 1 per cent of the full scale throughout the entire scale for the whole system (flowmeter, connecting cables, and recorder). For example, if the system is calibrated for a range of 0 to 5.6 liters, the reading will be correct to within 56 c.c. at any point on the scale. The reproducibility is plus or minus 0.5 per cent of full scale. The system is precalibrated and no further calibration previous to operating is necessary. No periodic maintenance is needed.

LABORATORY AND CLINICAL EVALUATION

This flowmeter is ideally suited for work with heparinized blood. Changes in blood constituents due to trauma are negligible.

To evaluate blood trauma from this unit, the following laboratory study was carried out. The flowmeter was placed in the line of 1.2 inch Tygon tubing, 1.75 meters in length, containing 280 c.c. of freshly drawn heparinized canine blood at near physiologic temperatures. Blood was circulated through this system by an occlusive roller-type pump at a flow of 3 liters per minute. Pressures within the system were monitored and held at 12 cm. of water. Samples for platelet count and plasma hemoglobin determination were drawn every half hour. At the end of 2 hours no fibrin deposits were encountered in the flowmeter. Differences in the plasma hemoglobin and platelet count, with or without the flowmeter in this system, were negligible. In this system the blood elements circulated through the flowmeter approximately 60 times as often as during a regular operative bypass period of 1 hour at 3 liter flow. Presumably the total trauma induced by the flowmeter during a surgical procedure would be proportionately less.

The flowmeter can be located anywhere in the extracorporeal system and can be positioned in the horizontal or vertical plane. It is preferable to have it after the arterial pump (Fig. 6). No presetting of the flowmeter or recorder is needed. As soon as the flowmeter is connected to the recorder it will give an accurate reading. The recording chart and integrator are operated only during the actual bypass. During bypass both continuous flow recording and cumulative volume readings are obtained. Mean flows are computed from this data.

Simultaneous recordings of mean arterial blood pressure, electroencephalographic patterns, venous pressure, and urine production, when related to flow, provide an accurate appraisal of the moment to moment physiologic state.

Prior to and after surgical bypass, when the pump oxygenator is "standing by," a recirculation shunt is used to prevent any portion of blood from standing in the flowmeter and overheating. Movement of less than one liter prevents deleterious changes in the blood from heat produced by the electromagnet.

The flow tube assembly, a rigid (Teflon) tube sheathed by a stainless steel jacket, is easily removed by unscrewing a knurled nut and pulling out the assembly. It is correctly replaced in the transmitter by aligning a notch on the

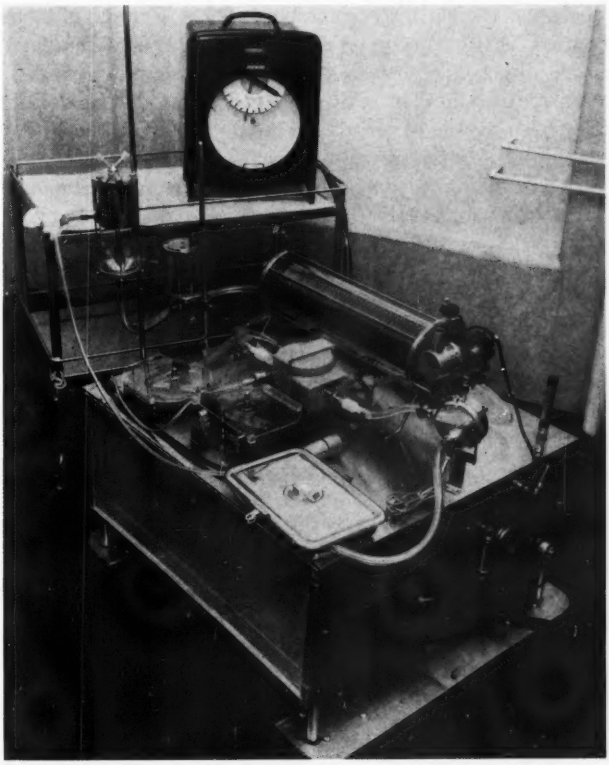

Fig. 6.—Transmitter and receiver assembled in the line of a pump oxygenator. Note transmitter is placed after the arterial pump but proximal to the filter.

jacket with a keying member. The flow tube is grounded at both ends to ensure that the blood is at the same potential throughout the tube. Ground contact is automatically made when the knurled nut is tightened.

The only meter component requiring sterilization is the removable flow tube through which the blood passes. Sterilization may be accomplished by ethylene

oxide gas, standard autoclaving or antibacterial fluids. End caps prevent contamination between sterilization and use. The two stainless steel "point" electrodes embedded in the flow tube tolerate sterilization.

This flowmeter has been in clinical use with our artificial heart-lung apparatus during the past 18 months. Its performance has been entirely satisfactory. It has also simplified the difficult problem of monitoring and recording blood flows in the laboratory.

SUMMARY

1. An electromagnetic flowmeter has been developed that meets important requirements in surgical and experimental extracorporeal circulation.

2. The operation of this instrument is based on Faraday's law of electromagnetic induction. Blood flowing through a magnetic field generates a voltage which is recorded directly in terms of flow in liters per minute.

3. The recorder is equipped with a direct writing pen that inscribes on a rotating chart. The system is preset and needs no calibration before use. A large dial provides instantaneous readings for the surgical team. An "integrator" gives a cumulative sum of the amount perfused during the entire bypass. The recorder can be placed more than 100 feet away from the flowmeter.

4. Accuracy is of the order of 1 per cent and is not altered by changes in density, turbulence, viscosity, temperature, pressure, conductivity of fluid, or by line voltage fluctuation up to 10 per cent.

5. There is no obstruction to flow or turbulence in the system. It does not traumatize blood. It is readily cleaned and sterilized.

6. The flowmeter and recorder have had extensive satisfactory clinical and laboratory use.

A NEW METHOD OF BYPASSING THE OBSTRUCTED VENA CAVA

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OBSTRUCTION of the superior vena cava occurs most commonly as a complication of intrathoracic diseases which have a fatal outcome. For this reason superior caval occlusion is usually managed by conservative measures. Nonoperative treatment is further justified because gradual improvement occurs naturally through the development of collateral circulation and may be enhanced by temporary regression of a neoplastic process following irradiation or chemotherapy. Furthermore, attempts to replace or bypass the superior vena cava with prostheses have been largely unsuccessful.

This report concerns a simple method of bypassing the obstructed vena cava with the greater saphenous vein.

CASE REPORT

E. A., a 52-year-old man, was admitted to Charity Hospital on May 8, 1959, complaining of continuous chest pain and edema of the face of 10 days' duration. During the 2 months preceding admission he had experienced exertional dyspnea and had lost 15 pounds.

On examination there was moderate periorbital and conjunctival edema, distended neck veins, and a 2 by 3 cm. mass in the right supraclavicular area. There were diminished breath sounds and dullness to percussion over the right upper chest. Urinalysis and blood count were within normal limits. Chest roentgenograms demonstrated a large mediastinal mass and consolidation of the right upper lobe. The right hemidiaphragm was paralyzed.

Venous pressure was 390 mm. of water in the right arm and 120 mm. of water in the right leg. The circulation time (arm-to-tongue) was 50 seconds. A venogram showed almost complete obstruction of the superior vena cava. Biopsy of the supraclavicular mass revealed poorly differentiated carcinoma. During July and August several courses of nitrogen mustard were administered with relief of chest pain but the manifestations of vena cava obstruction were unchanged.

On September 28 the patient was readmitted with marked progression of symptoms. Periorbital and conjunctival edema were pronounced, dyspnea was severe, and cyanosis was evident. Venous pressure was 450 mm. of water in the right arm and there was pronounced edema of both arms.

Operation was performed on Oct. 1, 1959. An incision was made over the greater saphenous vein from the ankle to the inguinal crease. The saphenous vein was ligated and divided at the medial malleolus. Beginning distally, all tributaries entering the saphenous vein were ligated and divided. As proximal dissection was carried out, heparin diluted

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in physiologic saline solution was injected into the vein to prevent deposition of fibrin. At the saphenous bulb the superficial epigastric and iliac circumflex veins were carefully protected since they were greatly enlarged and obviously serving as important collaterals for drainage of the superior vena cava. A subcutaneous tunnel was then constructed by making several 1 cm. transverse incisions in the skin of the anterior lateral region of the abdomen and thorax. A sponge forceps was passed between these incisions and into the upper end of the incision in the thigh. The distal end of the saphenous vein was pulled up through this subcutaneous tunnel taking care to avoid rotation or kinking. A short incision was then made over the external jugular vein at its junction with the subclavian vein. The neck incision was connected by blunt dissection to the uppermost thoracic incision, the distal end of the saphenous vein was brought into the neck, and an end-to-side anastomosis performed to the external jugular vein. No anticoagulants or antibiotics were administered after operation.

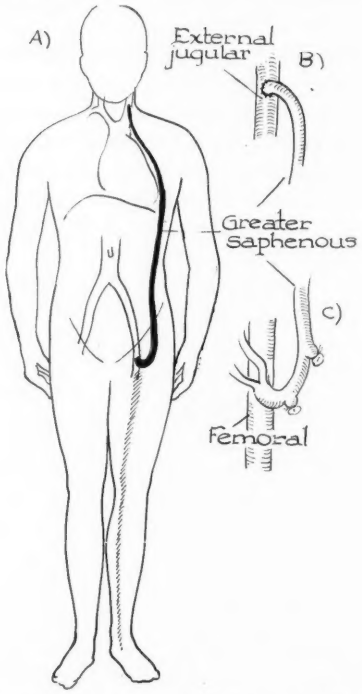

Fig. 1.—*A*, Diagrammatic representation of the operative procedure. The confluence of the saphenous and femoral veins is left intact. *B*, The distal saphenous vein is anastomosed end-to-side to the external jugular vein. *C*, Only the lowermost tributaries at the saphenous bulb are ligated. The superficial epigastric and iliac circumflex veins are preserved.

On the day after operation conjunctival edema was subsiding and there was marked relief of dyspnea. Periorbital and facial edema on the left side also decreased immediately and completely. On the right side of the face some edema persisted although it was greatly diminished. There was essentially no change in edema of the arms and hands until 4 weeks after operation when rather sudden decrease of edema occurred. This, too, was more pronounced on the left side. Concomitant with the changes in the edema of the upper part of the body the saphenous vein gradually dilated from a diameter of 0.4 cm. to 1.5 cm. Despite the improvement in the symptoms of superior vena cava obstruction, increasing weakness was apparent. On Nov. 12, 1959, the patient had massive hemoptysis and died. Permission for autopsy was not granted.

DISCUSSION

None of the previously described methods for operative relief of superior vena cava obstruction have proved to have wide applicability or long-term effectiveness. The procedure here described is readily tolerated and should be useful even in the presence of incurable conditions.

INTERNAL MAMMARY-CORONARY ARTERY ANASTOMOSIS

A Nonsuture Method Employing Tantalum Rings

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SURGICAL approaches to the treatment of occlusive coronary arteriosclerotic disease include (1) cardiopexy, using extracardiac structures to develop vascular adhesions to the myocardium, (2) arterialization of the coronary venous system, (3) implantation of the internal mammary artery into the myocardium, (4) ligation of the internal mammary artery to promote enlargement of pericardial branches communicating with cardiac branches at the base of the heart, and (5) coronary endarterectomy. None of these methods has been consistently successful.

Although it is generally true that systemic arteriosclerosis is a diffuse lesion, autopsy studies by Schlesinger and Zoll[1] have shown that coronary arteriosclerosis frequently results in segmental occlusion of a main stem. They have shown that in 63 per cent of 193 hearts studied, the occlusion measured 5 mm. in length or less and in 69 per cent of 125 hearts the proximal extent of the lesion was within the first four centimeters of the main coronary trunks. At this level the coronary arteries have a lumen of approximately 2.5 to 3.5 mm., a diameter that might permit direct systemic coronary arterial bypass anastomoses as a method of improving distal coronary circulation.

Direct suture anastomosis between systemic and coronary arteries has been attempted by other investigators with disappointing results,[2-9] even though the cannulated distal coronary arteries were perfused during the procedure to maintain coronary circulation. The incidence of early and late thromboses precludes clinical application of the method. Earlier work in this laboratory confirmed these findings and suture methods were abandoned. This report describes a nonsuture technique which employs a tantalum ring for the creation of internal mammary-coronary artery anastomoses.

MATERIALS AND METHOD

The rings used for the anastomosis are made of highly polished siliconized tantalum, 0.25 mm. thick, and vary from 2.5 to 6 mm. in length and from 2.5

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to 5 mm. in outer diameter. The rings are grooved circumferentially and there is a flange at the proximal end to facilitate handling with a hemostat (Fig. 1, *A*).

Mongrel dogs weighing 20 to 30 kilograms are anesthetized with intravenous Pentothal, intubated, and maintained on 100 per cent oxygen; supplemental Pentothal is given as required. The dog is placed in the right lateral position, the left chest entered through the fourth intercostal space and the left internal mammary artery mobilized from its origin down to the fifth intercostal space. To obtain adequate length in those situations where the artery is too narrow at this level, the branches of the brachiocephalic trunk are divided and later the internal mammary artery transected more proximally. The artery is not severed until the coronary vessel to be used is mobilized.

The pericardium is opened, the left atrial appendage retracted with a Satinsky clamp and either the left anterior descending or circumflex coronary artery, whichever is more accessible, is dissected for about 2 cm., care being taken to avoid the friable veins which may overlie the artery or receive tributaries across it.

Attention is now focused on the internal mammary artery. A bulldog clamp is applied proximally and the artery divided distally. The adventitia is dissected from the proximal cut end which then is passed through a ring chosen according to the size of the isolated coronary artery. The end of the artery is turned back over the ring as a cuff and fixed with a 4-0 silk ligature, tying it at the most proximal of the circular grooves while holding the flange of the ring with a hemostat (Fig. 1, *B*). It is important that the cuff be formed evenly to avoid the formation of a diaphragm across the lumen and that there be no rotation which would result in spiraling of blood flow. The bulldog clamp is momentarily released in order to check the adequacy of the blood stream.

The previously isolated coronary artery is now underrun with two 3-0 silk ligatures. The surgeon ties the proximal ligature only once, using a surgeon's knot. The first assistant then incises the vessel longitudinally, cutting from distal to proximal, by inserting the point of a bistoury-type blade and incising upward while the second assistant holds up the distally placed ligature to prevent back bleeding. The surgeon and the first assistant each pick up one edge of the cut vessel and then, as the surgeon inserts the cuffed internal mammary artery into the coronary artery, the assistant releases the distal ligature. This is then tied over the ring at the level of the most distal groove (Fig. 1, *C-H*). An intimal-to-intimal anastomosis results and dissection of blood between the coronary artery and the cuffed internal mammary artery is prevented, eliminating a possible site for later thrombosis. The bulldog clamp is removed and the anastomosis is functionally completed. The proximal ligature is now tied a second time and the coronary vessel between it and the ring divided (Figs. 1, *I* and 2). The pericardium is loosely closed, care being taken to avoid angulation of the anastomosis of the internal mammary artery. The wound is closed and both sides of the chest are drained for 24 hours. The time taken to perform

the anastomosis, measured from the moment of ligation of the coronary artery, has decreased with experience, the most recent attempts varying consistently from 34 to 60 seconds.

In all experiments the electrocardiogram and femoral artery pressure were recorded. Immediately after completion of the anastomosis an attempt was made to assess the adequacy of the anastomosis by temporary interruption of the internal mammary artery, monitoring the electrocardiogram and blood pressure.

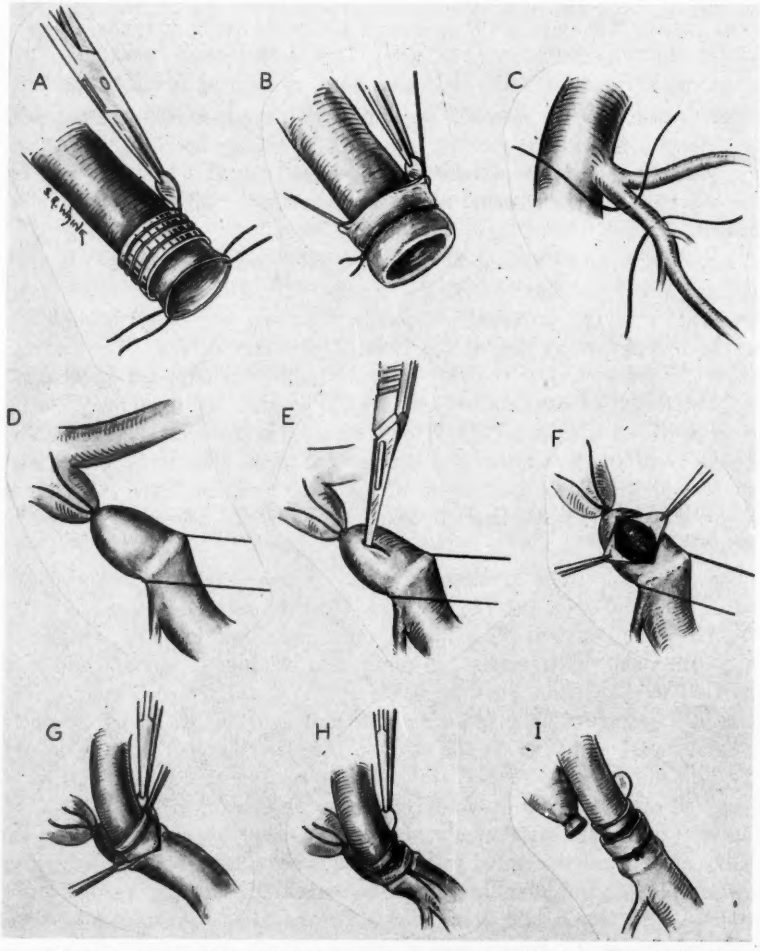


Fig. 1.—Technique of nonsuture coronary-mammary artery anastomosis. For explanation, see text.

Dogs failing to survive the procedure or dying in the early postoperative period because of infection were carefully studied by barium injection techniques to determine patency of the anastomoses (Fig. 3). The myocardium

was inspected for evidence of infarction and specimens were taken for histologic examination. The surviving dogs were subjected to coronary angiography at varying intervals and frequent electrocardiograms were obtained.

Fig. 2.—Completed anastomosis of left mammary artery to anterior descending branch of left coronary artery. Note cut end of proximal stump of coronary artery and Satinsky clamp retracting the left auricular appendage.

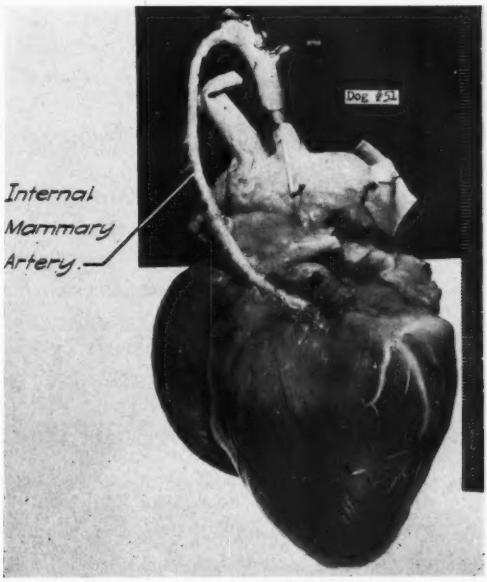


Fig. 3.—Injected specimen showing patency of anastomosis and absence of abnormal intercoronary anastomoses 6 months following anastomosis of left mammary artery to circumflex branch of left coronary artery.

Coronary angiography is performed using acetylcholine arrest as described by Arnulf in 1958.[10] A polyethylene cannula temporarily connected to a transducer is inserted via a femoral artery and advanced into the left ventricle. It is then slowly withdrawn into the ascending aorta, approximately 1 to 2 cm. above the aortic valve. In those cases in which the cannula does not readily enter the left ventricle, it is positioned by fluoroscopy. Three to five milligrams of freshly prepared acetylcholine is injected via a peripheral vein and, when cardiac arrest is identified by cessation of electrocardiographic activity and disappearance of the monitored pressure in the saphenous artery, 10 c.c. of contrast medium is injected by hand. Using a rapid film changing apparatus designed by one of us (R. H. G.), films are exposed at the rate of two per second starting after approximately 5 seconds of arrest and continuing through the first three or four heart beats after cessation of arrest. The duration of arrest ranged from 10 to 20 seconds and all of the animals recovered promptly with no untoward sequelae. The procedure was repeated, in some instances as many as four time, where indicated, without complication. In 1 animal the cannula perforated the aorta and the animal died from cardiac tamponade.

RESULTS

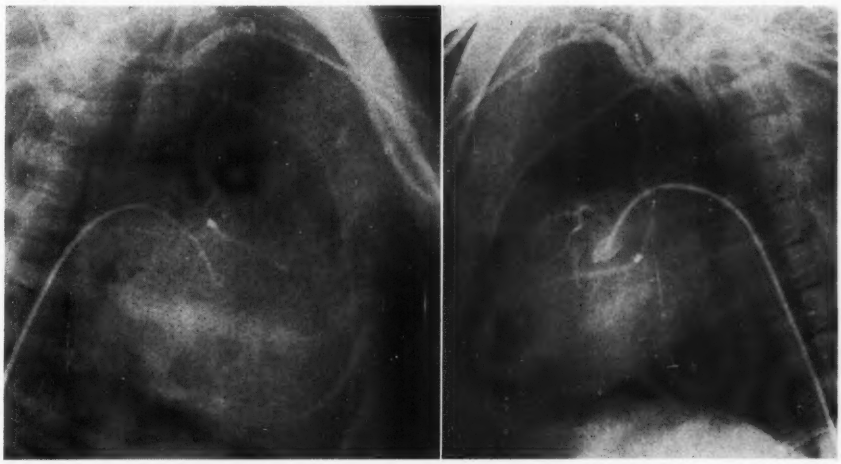
Many methods were employed in an effort to perform the anastomosis rapidly and protect the myocardium from irreversible arrhythmias. Hypothermia was used and abandoned because, while it significantly slowed the heart and appeared to provide a longer safe period for the procedure, a number of the animals died as a result of irreversible ventricular fibrillation. When the definitive procedure was demonstrated to be satisfactory, a group of 12 dogs was operated on in sequence. Three of the animals, after having recovered successfully from anastomosis of one of the branches of the left main coronary artery, underwent internal mammary-coronary artery anastomosis of the remaining branch, 1 to 3 months later. There were thus fifteen procedures completed in the 12 animals that comprise this report.

One dog died as a result of irreversible ventricular fibrillation. Two animals died on the third and fourth postoperative days, respectively, of atelectasis and pneumonitis. One dog survived 110 days after the first anastomosis but died 13 days after the second anastomosis when the aorta was, as already mentioned, perforated during coronary angiography. Another dog died on the eighteenth postoperative day of pneumonia. Thus 5 animals died after creation of six anastomoses (one surviving 110 days before the second procedure), 4 from causes not related to the procedure. Five of the six anastomoses were patent on autopsy study. No evidence of myocardial infarction was observed in any of these animals.

The remaining 7 dogs have been classified as long-term survivors. Three animals lived for a minimum of 6 months before they were sacrificed and the remaining 4 are alive from 7 to 20 months to date. Nine anastomoses were completed in the 7 dogs, eight of which were demonstrated to be patent either at

time of necropsy or by coronary angiography. The animals have apparently been in good health with no electrocardiographic alterations and seem to have a normal exercise tolerance.

Thus, of the fifteen anastomoses completed, only two were demonstrated to have thrombosed, although the time interval in five of the procedures was too short to be conclusive. However, it is becoming increasingly apparent that if the anastomoses remain patent for at least 1 week after operation that late obstructions are unlikely to develop. Two typical examples of a patent anastomosis are illustrated in Fig. 4. Some inferential evidence that flow through the anastomoses is adequate is provided by postmortem barium injection of the heart. It is well known that chronic ischemia is a powerful stimulus for the development of intercoronary anastomoses. The absence of such anastomoses (Fig. 3) suggests that coronary flow was satisfactory.



A. *B.*

Fig. 4.—*A*, Coronary arteriogram demonstrating patency of mammary to right coronary artery anastomosis.

B, Coronary arteriogram demonstrating patency of anastomosis between mammary and left anterior descending coronary arteries.

DISCUSSION

It has become apparent that suture techniques for creating systemic artery-coronary artery anastomoses result in such a high percentage of occlusions as to make their clinical application unsuitable. A nonsuture technique similar to ours, using polyethylene rings, has been described by Carter and Roth[11] and appears to result in a higher proportion of long-term patency. The ring technique permits more rapid anastomosis within a safe period of myocardial ischemia in the normal canine heart.

The mortality rate following acute ligation of the left anterior descending coronary or left circumflex coronary artery, as reported in the literature, is high but variable. McEachern and co-workers[12] found that 25 per cent of dogs

survived ligation of the left circumflex and 60 per cent survived ligation of the left anterior descending artery. Other investigators report survival rates for left anterior descending artery ligation of 30 per cent[13] and 72 per cent.[14] The discrepancy in results is related to the site of origin of the anterior septal branch. When this arises from the left anterior descending artery and is obstructed by ligation, the mortality rate is markedly increased.

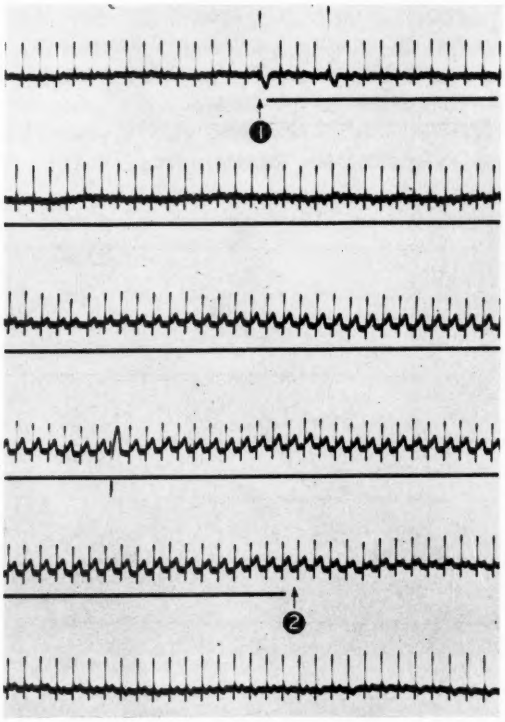


Fig. 5.—Electrocardiogram recorded during anastomosis of left mammary artery to circumflex branch of left coronary artery. At *1* coronary artery occluded. At *2* anastomosis completed and clamp on mammary artery released. Time of interruption of circulation 53 seconds. Note rapid return to normal.

During the experimental study, the electrocardiogram was monitored and changes reflecting myocardial ischemia developed during the period of temporary coronary occlusion in all studies. These changes reverted to the preocclusion pattern upon completion of the anastomosis, suggesting adequacy of coronary blood flow through the anastomosis (Fig. 5).

The use of polished siliconized tantalum for the procedure was suggested by successful application of the method in a study of small artery anastomoses in this laboratory. It was observed that arteries as narrow as 2 mm. in internal diameter remain patent if proper care is taken during the operative manipulations. The method is much more simple than direct suturing of these small

friable vessels and eliminates the direct contact of a foreign body with the intima and blood stream, a factor which may be important in the development of thromboses.

Gross studies of the sacrificed animals in this series reveal considerable tissue reaction in the areas of dissection, a firm intimal union between the mammary and coronary vessels and minimal reactivity between the arteries and the tantalum ring.

The clinical applicability of the procedure remains conjectural. Certainly the autopsy material studied by others[1] would indicate that a significant number of patients with coronary arteriosclerosis have segmental obstruction in the proximal portions of the main coronary arteries. Many such patients suffer from frequent attacks of angina pectoris, and some are severely incapacitated. The myocardium in such situations is unsatisfactorily maintained by a collateral circulation which is in constant jeopardy of ultimate obstruction. It would thus appear reasonable to bypass the area of obstruction with some form of systemic coronary arterial anastomosis. The very fact that the main coronary artery is segmentally obstructed would tend to add greater security in performing the procedure since not even temporary occlusion of the proximal end would be required, further myocardial ischemia should not occur and, with a patent distal coronary system, a more satisfactory perfusion of the myocardium should result. The choice of suitable patients for the procedure, unfortunately, cannot be dictated by the clinical status but must rest upon satisfactory methods of preoperative roentgenographic visualization of the coronary arteries.

Techniques for satisfactory coronary angiography have been demonstrated[10, 15-18] and are being further developed. The use of acetylcholine arrest in this laboratory has proved satisfactory in dogs and excellent visualization of the coronary arteries has been possible.

CONCLUSION

Attempts to create systemic-coronary artery anastomoses using suture techniques have not been satisfactory in this laboratory or in the experience of other investigators. Early thromboses occur very frequently, and long-term patency cannot be predicted with any degree of certainty. A nonsuture method using tantalum rings has been discussed and the results of fifteen consecutive anastomoses between the internal mammary artery and either the left anterior descending or circumflex artery have been presented. Thirteen of the anastomoses were demonstrated to be patent (1) at the time of necropsy if the animal succumbed postoperatively, (2) when sacrificed after at least a 6-month survival, and (3) by coronary angiography using acetylcholine cardioplegia. The longest survival to date has been 20 months and patency has been demonstrated by coronary angiography.

The results using nonsuture techniques are superior to direct suture methods and long-term patency appears to be significantly more predictable in normal dogs. The possible clinical application of the procedure to bypass areas of

segmental coronary artery occlusion has been suggested. Further study is continuing in the hope that it will prove feasible to add this method to the surgical armamentarium in the treatment of coronary arteriosclerosis.

The authors are indebted to Mrs. S. Selmonosky and Mr. A. Leon for their technical assistance, to Mr. Stanley Waine for the drawings, and to Mr. Milton Kurtz for the photography. Drugs and fluids were kindly supplied by Abbott Laboratories, North Chicago, Ill., and suture materials by Deknatel and Son, Inc., Queens Village, N. Y.

ADDENDUM

Since this article was submitted for publication, a coronary mammary anastomosis between the right mammary and right coronary artery, using the tantalum ring, has been successfully performed on a 38-year-old patient at Van Etten Hospital, Bronx Municipal Hospital Center, New York, on May 2, 1960. The patient is doing well and it is intended to report the case in detail in a separate communication.

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INDUCED CARDIAC ARREST

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In our laboratory we have found it comparatively easy to achieve survival of dogs after 30 minutes of total body perfusion with the use of a pump-oxygenator. Of ten perfusion experiments with the spinning disc oxygenator, six were "long-term," that is, of more than 1 week's survival. When cardiac arrest was added to the perfusion, the mortality rate increased. It was therefore evident that cardiac arrest per se is a significant factor in the survival.

Helmsworth and his associates[1] found in their experimental work on dogs that total body perfusion caused some myocardial damage. They observed fatty degeneration of mild degree and, in rare foci, frank necrobiosis. These changes could only be observed after a survival of more than 6 hours. In 3 cases the survival time was 48 hours. When asystole also was induced by potassium, a serious degree of myocardial necrosis was observed in 4 cases of 48 hours' survival. In these experiments they used a method for drainage of the blood from the left heart during the perfusion. When Helmsworth's group compared the anoxic with the potassium arrest, the same degree of fatty degeneration of the myocardium was found but focal necrosis was absent or very slight when anoxic arrest was used. They concluded that a short period of anoxic arrest was superior to potassium arrest. The heart-lung bypass lasted for 30 minutes in all their experiments. Löhr and Gillmann[4] on the other hand found a higher incidence of pathologic changes in the electrocardiogram after anoxic than after the potassium arrest. Only 6 to 10 minutes of occlusion, however, were used.

We have undertaken an experimental study in order to compare the heart reaction to arrest induced by anoxia, potassium, acetylcholine, or deep hypothermia.

METHODS

A spinning disc oxygenator with a De Bakey roller pump and gravity drainage was utilized on dogs. Both venae cavae, the left atrium, and the femoral artery were cannulated in the ordinary way. Anoxic cardiac arrest was induced by applying a clamp over the root of the aorta. Potassium arrest was produced according to the Melrose technique[5] and acetylcholine arrest according to that of Lam.[2] Arrest was continued for 30 to 40 minutes in all experiments. It was necessary to sacrifice 5 to 7 dogs for each experiment to obtain enough blood for the perfusion and the postoperative treatment.

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In the series of dogs under deep hypothermia, the blood from the left atrium was drained into a reservoir and pumped through a heat exchanger and a filter back into the femoral artery. When ventricular fibrillation occurred, which usually happened at a temperature of about 20° C. in the esophagus, the machine was stopped while a catheter was pushed into the pulmonary artery through a stab wound in the right ventricular wall. The blood from the right atrium was then drained to another reservoir, pumped into the pulmonary artery, oxygenated in the lungs, returned to the left atrium and pumped through the heat exchanger back to the femoral artery. The temperature of the experimental animal on total body perfusion was lowered to about 10° C. in the esophagus. All circulation was then stopped and hypothermic arrest was maintained for 30 to 40 minutes. Rewarming was achieved in the same manner, utilizing two pumps, until the heart recovered a regular beat, spontaneously or after defibrillation. Thereafter only the left-sided circuit was continued for rewarming.

In all experiments, the animals which survived more than 4 hours were either sacrificed or autopsied immediately after death at which time specimens from the anterior wall of the left ventricle was removed for histopathologic investigation. These specimens were always taken at the same site of the heart for the sake of comparison. One piece was placed in absolute alcohol and another was placed in 10 per cent formalin. For glycogen, Best's carmine stain was used. Fat stainings were performed with Sudan III or scharlach R. The other stains used were hematoxylin-eosin, van Gieson–hematoxylin, and, in some cases, periodic acid reaction according to MacManus or Mallory's phosphotungstic acid hematoxylin. In 2 cases, the (malic) dehydrogenase reaction, according to Neoral and co-workers,[6] was performed and the dark staining of the myocardium by the reagent was compared with that obtained on the healthy myocardium of a blood-donor dog which had been sacrificed.

RESULTS

In a series of fifteen experiments, four of anoxic cardiac arrest and eleven of potassium arrest, we obtained only one "long-term" survival. This case was in the group in which potassium was used. Thus it was impossible to use survival time as a criterion to differentiate between the potassium and the anoxic groups in our laboratory where many dogs die during the night after the perfusion because of inadequate blood replacement.

In a second series of thirty experiments, we produced different types of cardiac arrest and studied the myocardium histologically. Twelve of the dogs survived for more than 6 hours. The longest survival time was 27 hours. To obtain 30 to 40 minutes of arrest using anoxia, potassium, or acetylcholine, a total perfusion time of about 60 minutes was used, with the longest perfusion time being 220 minutes. A longer time of perfusion was needed to obtain a cardiac arrest for 30 to 40 minutes using deep hypothermia, an average of 110 minutes. In these experiments it was observed that a severe tricuspid insufficiency occurred in 2 cases in that group in which anoxic arrest was performed (Fig. 1). In the first case the insufficiency appeared after 35 minutes

of arrest and further perfusion was needed for 55 minutes to take the work load away from the heart in order to correct the insufficiency. In the second experiment, the tricuspid insufficiency appeared after 30 minutes of arrest but only 20 minutes more of perfusion were needed before the insufficiency vanished. No such insufficiency could be observed in the other groups.

The changes in the myocardium of the dogs that survived for 4 hours were very small and appeared only as a diminished glycogen content and an interstitial edema. There were no significant changes between the different types of cardiac arrest.

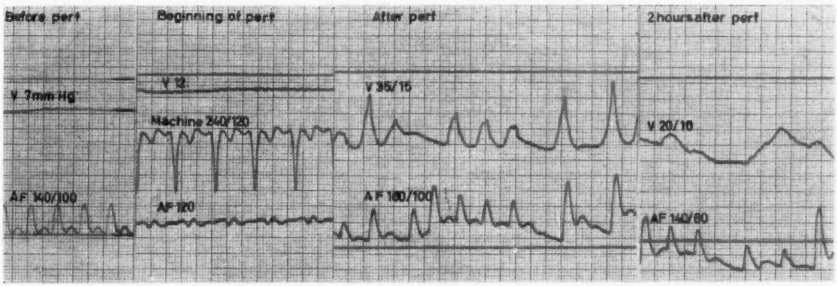

Fig. 1.—Anoxic arrest for 35 minutes. The pressure in the vena cava *(V)* was 7 mm. Hg before and 12 mm. Hg during perfusion. After perfusion there was a pronounced tricuspid insufficiency with a vena cava pressure of 35/15 mm. Hg necessitating an additional 33 minutes of perfusion. The tricuspid insufficiency then diminished and 2 hours after the perfusion the vena cava pressure was 20/10 mm. Hg.

The myocardial changes in the dogs that survived for 6 hours or more are summarized in Table I. The most severe changes were found in the group having had potassium arrest. There was loss of the glycogen content. In one case we found a marked fatty degeneration (Fig. 2). Degenerative changes of Zenker's type were prominent with practically complete loss of striation and pronounced fiber swelling (Figs. 4 and 7). In some places granular degeneration and, in a few foci, frank necrosis with sparse inflammatory cells were seen (Fig. 6). Interstitial edema and hemorrhage were prominent. The next severe changes were registered in the group with anoxic arrest (Fig. 8). The glycogen content was a little diminished. Fat-stained slides presented some red pigment granules at the poles of the nuclei of the type "normally" found but no fat droplets were seen in the rest of the muscle thread. Small necrobiotic changes, interstitial edema and, in a few places, granular degeneration were observed in the other stainings. Only very small changes were found in the acetylcholine (Fig. 9) and the hypothermic groups (Figs. 5 and 10). However, some changes were noted in 2 dogs, No. 41 and No. 44, of the hypothermic group (Table I and Fig. 3). In both these experiments some technical errors were made during the perfusion causing a diminished perfusion flow and a severe metabolic acidosis. The pH decreased to 7.1 in experiment 41 and to 7.24 in experiment 44 with a simultaneous decrease of the bicarbonate down to 12 and 15 mEq., respectively.

Fig. 2.

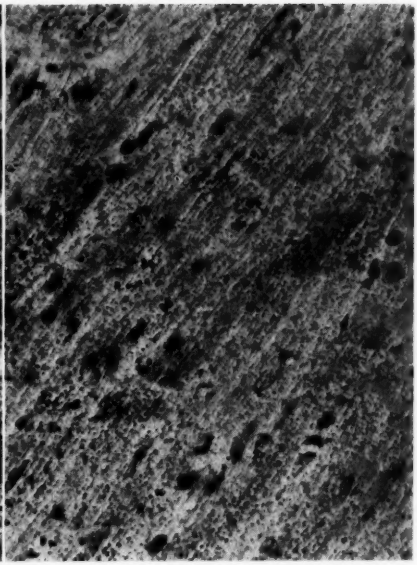

Fig. 3.

Fig. 2.—Exper. 33. Potassium arrest. Fat staining (scharlach R). This photomicrograph demonstrates highly pronounced fatty degeneration of the myocardial threads (fat droplets dark). (×620; reduced 2/7.)

Fig. 3.—Exper. 44. Hypothermic arrest. Fat staining (scharlach R). Small fatty deposits in the myocardium are shown. (×620; reduced 2/7.)

In experiments 44 and 45 of the hypothermic group, the dehydrogenase reaction was performed. No evident changes were found.

TABLE I

	ANOXIA			POTASSIUM		ACETYL-CHOLINE		DEEP HYPOTHERMIA				
								16.7° C.	9.5° C.	10.4° C.	10.0° C.	9.5° C.
No. of experiments	29	35	39	33	37	31	26	40	41	45	44	43
Duration of arrest	30	35	30	35	30	35	30	40	30	40	30	30
Duration of perfusion (min.)	57	55	60	55	50	67	55	145	70	129	104	67
Survival time (hours)	6	6	6	6	10	6	7	6	7	10	12	27
Glycogen content	+	++	++	−	+	+	+++	++	++	+++	(+)	+++
Fatty degeneration	−	−	−	+++	−	−	(+)	−	+	−	++	−
Impaired striation and staining properties	++	+	+	+++	++	−	−	−	+	−	−	−
Fiber swelling	−	+	+	++	++	−	−	−	+	−	−	−
Granular degeneration	+	+	−	++	+	−	−	−	+	−	−	−
Necrosis	−	−	−	+	+	−	−	−	−	−	−	−
Cellular infiltration	−	−	−	+	+	−	−	−	−	−	−	−
Interstitial edema	++	+	+	++	++	−	−	−	−	−	+	+
Interstitial hemorrhage	−*	−	−†	+‡	+‡	−	−*	−	+	+	+	−

*Right ventricular dilatation.

†Postmortem changes cannot be excluded.

‡Right and left ventricular dilatation.

Fig. 4.

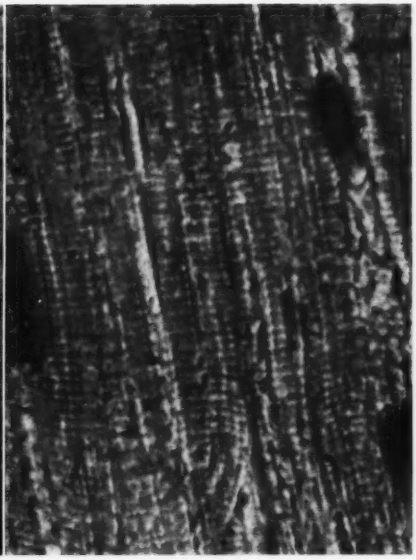

Fig. 5.

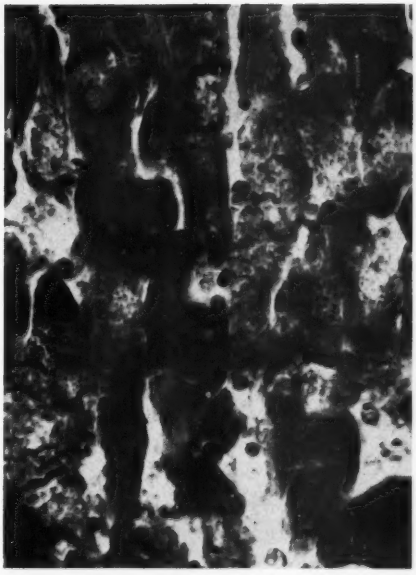

Fig. 6.

Fig. 4.—Exper. 33. Potassium arrest (van Gieson-hematoxylin). Photomicrograph which demonstrates marked loss of striation and fiber swelling. (×2,300; reduced 2/7.)

Fig. 5.—Exper. 40. Hypothermic arrest (van Gieson-hematoxylin). This photomicrograph demonstrates well-preserved striation and architecture of the myocardium. (×2,300; reduced 2/7.)

Fig. 6.—Exper. 33. Potassium arrest (phosphotungstic acid hematoxylin). This photomicrograph demonstrates pronounced fiber swelling, practically complete loss of striation, and necrosis with single interstitial granulocytes. (×980; reduced 2/7.)

Fig. 7.

Fig. 8.

Fig. 9.

Fig. 10.

Fig. 7.—Exper. 33. Potassium arrest (phosphotungstic acid-hematoxylin). This photomicrograph demonstrates fiber swelling, pronounced loss of striation, and granular degeneration. (×980; reduced 2/7.)

Fig. 8.—Exper. 29. Anoxic arrest (phosphotungstic acid-hematoxylin). Areas with loss of striation and granular degeneration are shown. (×980; reduced 2/7.)

Fig. 9.—Exper. 26. Acetylcholine (phosphotungstic acid-hematoxylin). A rather well-preserved striation is shown. (×980; reduced 2/7.)

Fig. 10.—Exper. 40. Hypothermic arrest (phosphotungstic acid-hematoxylin). A well-preserved striation is shown. (×980; reduced 2/7.)

COMMENTS

Cardiac arrest induced by anoxia may obviously produce enough changes in the myocardium to cause a tricuspid insufficiency; but this insufficiency is reversible after a prolonged period of total body perfusion (Fig. 1). No such incidence of tricuspid insufficiency was noted during potassium, acetylcholine, or deep hypothermic arrest. To discriminate against the anoxic method on this basis is unjustified because of its reversibility, but it corresponds to the findings of Löhr presented above.

It is generally agreed that 6 hours of continuous ischemia must elapse before any necrobiotic changes of the myocardium are observed in hematoxylin-eosin stained slides. In dogs in which ischemia was produced by occlusion of a vessel, Neoral and co-workers[6] found a diminished glycogen content after 1 hour and a decreased dehydrogenase reaction and a fatty degeneration after 5 hours of ischemia. Our findings are similar: at least 6 hours must elapse before significant changes become evident with our methods. In our experiments, however, the ischemia is only maintained for a comparatively short period. It is, therefore, possible that necrobiotic changes may appear even later when debilitated cells surrender, but our observations do not point in this direction.

The different histologic changes, found in the myocardium, are of different importance. A partial loss of the glycogen content is probably only a sign of exhaustion and certainly reversible. Fatty degeneration precedes the more pronounced alterations, and this also seems to be reversible. In our group of dogs having had anoxic arrest there was no fatty degeneration but necrobiosis. Here the degeneration had possibly started very abrubtly and the state of fatty degeneration bypassed, with only frank necrobiosis appearing. The interstitial edema and hemorrhage, if not pronounced, is probably transitory and, at least to some extent, an effect of the operative procedure. The changes found in the groups in which cardiac arrest was induced by acetylcholine or deep hypothermia are reversible (except experiment 41).

The number of "long-term" survivors after total body perfusion and induced cardiac arrest is small. It may, therefore, be thought a little hazardous to institute comparisons between the different methods to induce the cardiac arrest. However, the morphologic investigations were performed independently and without knowledge of the method used to produce arrest or of the survival time. The metabolic acidosis of the dogs No. 41 and No. 44 in the group in which arrest was induced by deep hypothermia is well reflected in the myocardium. In the potassium group, there are definite pathologic changes in the myocardium of both the dogs (see Table I). These facts give us the right to draw a comparison between the different groups. If deep hypothermia or acetylcholine arrest is performed without metabolic acidosis, smaller changes of the myocardium are observed than in anoxic or potassium arrest. The potassium method seems to be a rather dangerous and doubtful one. This last statement agrees with the findings of Helmsworth and his group. Considering the deep hypothermia and the acetylcholine methods we think the former to be the best

for the myocardium, since by this method one gets a longer cardiac arrest without danger, thanks to the strongly diminished oxygen demands of the myocardium at the lower temperature. Our histologic investigations did not include the brain. There were, however, no "clinical" signs of functional change in the central nervous system. The oxygen supply for the brain must be considered as well as for the myocardium, if the method is to be used in operative procedures upon human beings.

SUMMARY

An experimental study has been performed on dogs to compare the different methods of inducing cardiac arrest. The arrest was maintained for 30 to 40 minutes. Pronounced pathologic changes in the myocardium were observed after potassium arrest. Smaller changes were seen after arrest produced by anoxia and very little alteration was found after hypothermic and acetylcholine arrest. The deep hypothermic method is to be preferred because of the diminished oxygen demands at the lower temperature during this type of cardiac arrest.

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THE MEASUREMENT OF BLOOD FLOW THROUGH EXTRACORPOREAL CIRCUITS

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THE PRIMARY function of an artificial heart-lung machine is to provide the body with a necessary amount of oxygenated blood during cardiopulmonary bypass. It seems evident that the most important information of the performance of artificial circulation is the blood flow itself.[1-5, 7-9, 15-18] Despite the recognition of this fact, the majority of surgical teams have only a rough estimate of the blood flow through the extracorporeal circuit and do not follow, control, or record changes in the flow rate which may occur during perfusion. Without this information the operative hazard is increased, and valuable physiologic information is lost.

The need for a reliable flowmeter is generally recognized. A cross section of research work in this field was recently presented on a special Symposium at Omaha, June 17-19, 1959, where the following criteria were established for an ideal blood flowmeter.[16] (1) The instrument must be rugged, record accurately, and the calibration curve and base line should remain stable. (2) It should not tend to produce clots or hemolysis, more than any part of the system. (3) It should not alter the blood flow characteristics or interfere with the pump function. (4) It should be instantaneously responsive, and not be affected by the pulsatile flow, changes in temperature, viscosity, and density within physiologic ranges. (5) Installation and cleaning should be easy and operation simple. (6) Signals should be readable on available recording instruments for both flow rate and total flow.

However, most of the research work is concentrated on the problem of how to measure flow inside rather than outside blood vessels. Several methods were described for the measurement of blood flow through extracorporeal circuits. Some of these methods ("calibration of the pump," temporary occlusion of the circuit, etc.) proved to be inaccurate; others (electromagneticity, ultrasound, radioactivity, etc.) are still in the experimental stage and are not generally accepted in the clinical practice. In this paper we wish to give a review of methods used at the Charlotte Memorial Hospital designed to measure the flow

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through the pump oxygenator. For this purpose we used two different kinds of flowmeters: one which measures the flow through the arterial (return) line of the circuit, the other for determining the gravity venous discharge.

FLOWMETER I

Our first flowmeter, the *orifice-plate*[10, 11] belongs to the group of fluid meters specified as head (kinetic) flowmeters. Its principle is based on the hydraulic law, that fluid under pressure gains velocity and loses pressure-head when passing through a stricture. The secondary element—a special recording manometer—uses this pressure differential to infer the rate of flow. The orifice-plate is one of the oldest devices for measuring flow of liquids. Records indicate that Romans in Caesar's time used this method to measure water going to residences, but only in the last decade has this instrument for measuring flow in pipes been systematically studied.

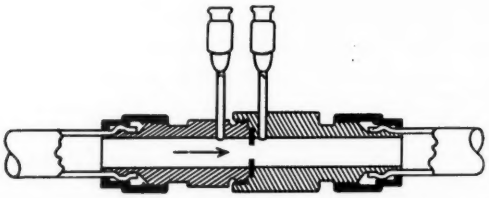

Fig. 1.—The orifice-plate flowmeter (schematic drawing).

The steady flow general energy equation between points 1 and 2 may be written in the following manner:

$$z_1 + u_1 + p_1v_1 + \frac{V_1^2}{2g} + Q + W = z_2 + u_2 + p_2v_2 + \frac{V_2^2}{2g}$$

in which:

v_1v_2 = specific volume of fluid at respective points.
z_1z_2 = elevation between points 1 and 2.
V_1V_2 = velocity at points 1 and 2.
u_1u_2 = internal energy of fluid at points 1 and 2.
W = the net amount of work performed.
Q = the net heat transfer.

In most cases, as in the present case, the z,Q,u, and W terms may cancel, and the equation may be written in the following form:

$$p_1v_1 + \frac{V_1^2}{2g} = p_2v_2 + \frac{V_2^2}{2g}.$$

Replacing the specific volume v by its reciprocal specific weight y and expressing $V_1 = V_2 \frac{D_2^4}{D_1^4}$ for conduits of circular cross-section D, the equation will be:

$$V_2 = \frac{1}{\left(1 - \frac{D_2^4}{D_1^4}\right)^{1/2}} \sqrt{2g \frac{p_1 - p_2}{y}}$$

where the expression $\frac{1}{\left(1 - \frac{D_2^4}{D_1^4}\right)^{1/2}}$ is known as the velocity approach factor and may be represented by the letter M:

$$V_2 = M \sqrt{\frac{p_1 - p_2}{y}}.$$

The volumetric rate of flow is equal to the area of the fluid stream A multiplied by the average velocity V:

$$F = AV = AM \sqrt{2g \frac{p_1 - p_2}{y}}.$$

Because for a given fluid and flowmeter the factors A, M, and y are constant C, the flow can be expressed by the difference of pressure between the points 1 and 2 of the meter:

$$F = C(p_1 - p_2).$$

The constant C may be calculated or gained empirically.

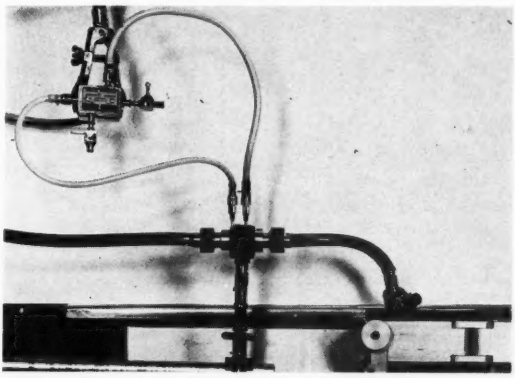

Fig. 2.—The orifice-plate flowmeter with the differential pressure transducer in the arterial (return) line of the extracorporeal circuit.

Most of the devices used for physiologic pressure measurements can be used as a secondary element to measure the pressure differential between the two pressure taps. In the beginning we applied two separate Statham 23B pressure transducers, but lately we have used a single Sanborn 267B-2 differential pressure transducer. The monitoring and recording are done with a multichannel oscillograph, which also allows the integration of the flow curve. By empirical calibration this pressure differential can be directly translated into a flow of cubic centimeters per minute.

In the last two years this flowmeter has been used routinely in our open-heart operations[10, 11, 13, 14] to measure the flow through the arterial line of the pump-oxygenator. This instrument is 3 inches long, weighs less than 300 grams, and is made from silicone-coated stainless steel. The plate itself is Teflon and is changeable. The flowmeter is provided with Luer-lok pressure taps on either side of the plate and has safety connectors on both ends for the plastic tubing.

The common disadvantage of all kinetic meters is that the acceleration and deceleration of the blood stream causes some degree of injury to the blood. This is somewhat higher in the orifice-plate than in the Venturi-tube, which was also used as a blood flowmeter.[6, 12] On the other hand, for the same diameter ratio the pressure gradient (sensitivity of the meter) is much higher for the orifice-plate than with the Venturi-tube. In other words, the narrowing of the circuit and the proximal pressure build-up are much higher for a Venturi-tube than for an orifice-plate of the same sensitivity. By using a sensitive recording device and a relatively large orifice, the turbulence and hemolysis caused by an orifice-plate flowmeter should not exceed that in a moderate pulmonary stenosis.

The free hemoglobin content of the plasma as an index of blood injury was also routinely determined in all of our patients after open-heart surgery. With the flowmeter in the described extracorporeal system, following perfusions from 9 to 45 minutes, the free hemoglobin content of the plasma was less than 130 mg. per 100 ml. This does not exceed the average values found in literature.

To study the pumping efficiency and the hemolysis, blood was recirculated through an extracorporeal system composed of a rotating disc oxygenator, heating unit, twin rotor pumps, gravity reservoir, filter and bubble trap. Using identical mixtures of donor blood, the perfusion was repeated with an orifice-plate flowmeter in the system. Comparing the results of the experiments, we found the free hemoglobin content of the plasma to be practically the same. The difference in perfusion pressure/flow ration was 0.03.

FLOWMETER II

Our second flowmeter is especially designed for *gravity venous drainage,* but it can be used anywhere in an extracorporeal circuit where the function allows free fluid surface and discharge under atmospheric pressure. Like the orifice-plate, this instrument also belongs to the group of "variable head meters," but because of the free discharge the flow is expressed in pressure head, rather than pressure differential. To build up such a pressure head, the flowmeter utilizes a constriction caused by a simple outflow nozzle.

The formula for discharge through such a contracted opening is the same as that for a Venturi flume:

$$F = CMa_2\sqrt{h}$$

where

$$M = \sqrt{\frac{2g}{1 - \left(\frac{a_2}{a_1}\right)^2}}$$

In the above expressions h is the drop in fluid surface, a_1 and a_2 are, respectively, the effective cross-sectional areas of the stream just above the pronounced drop in the fluid surface and the area of the smallest cross-section of the opening. The coefficient C corrects for "minor losses," e.g., friction, contraction and turbulence. With proper design these losses could be minimized.

The primary element of the flowmeter consists of two communicating siliconized glass tubes. The blood enters through a low resistance orifice, located

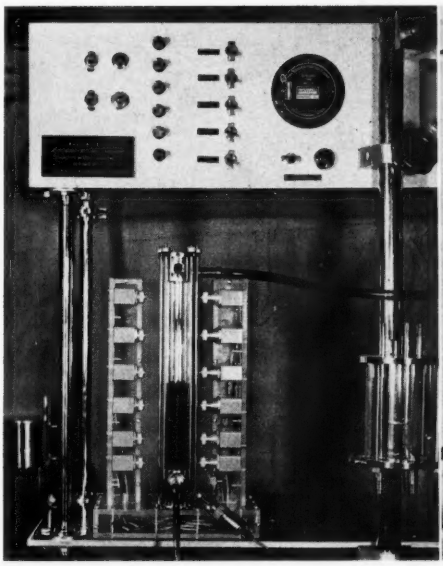

Fig. 3.

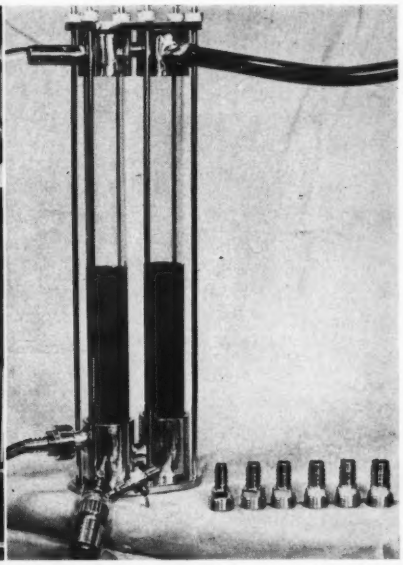

Fig. 4.

Fig. 3.—The gravity discharge meter without the tertiary element.
Fig. 4.—The gravity discharge meter with the tertiary element and monitoring panel.

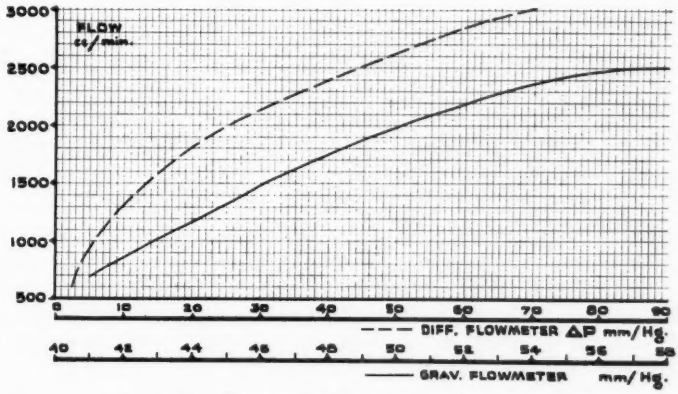

Fig. 5.—The flow characteristics of the two flowmeters.

at the upper end of the first (inflow) tube and leaves through a contracted outflow nozzle. Because of the impeded flow—outflow—there is always a level of blood in the metering tube. At extreme flow levels, the blood is freely discharged through the upper end on the metering tube.

The height of the blood column (the pressure head), according to the principles described, will be proportional with the actual flow rate. This proportion is determined by empirical calibration and marked on the outer surface of the metering tube, allowing instantaneous reading of the flow.

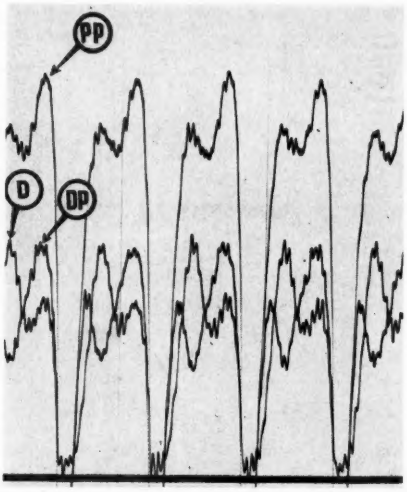

Fig. 6.—The original, undamped pressure curves of the orifice-plate flowmeter; *PP*, proximal pressure tap; *DP*, distal pressure tap; *D*, differential pressure.

The secondary element of the flowmeter allows distant observation and recording. Any of the commercial pressure monitoring devices, already available in operating rooms, could be used for this purpose. We use a 23B Statham pressure transducer to measure the pressure head (static pressure of the blood column in the metering tube). The recorded curve is directly calibrated in a flow of cubic centimeters per minute.

Recently we have used an additional secondary (may call it tertiary) element which is not in actual contact with the flowmeter. It consists of a series of photoelectric cells and 8 volt light bulbs which are symmetrically mounted at the two sides of the meter. The light bulbs—through the metering tube of the flowmeter—activate the photoelectric cells. The increase in flow raises the blood level in the meter and covers the light bulb at the respective level. The inactivation of the corresponding photocell relays special light and sound signals to the instrument panel of the pump-oxygenator. These signals could be calibrated with a desired range and accuracy. The device is set to give special flickering light signals at the dangerously low or too high flow rates.

The advantage of our gravity outflow meter is its absolute reliability. The primary device consists of a few stainless steel and glass parts and the working

principle is based on a simple physical law. The metering process does not involve the function of electric or electronic devices; it is independent of the function of the pump, temperature, size, and material of the tubing and cannulas, and is influenced by a single factor—the blood flow itself. This practically excludes the possibility of a mechanical failure or inaccuracies.

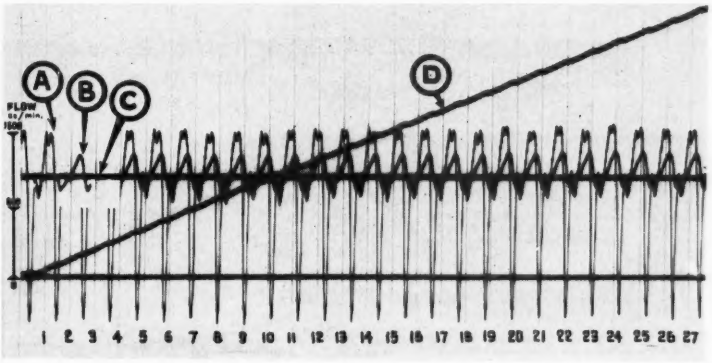

Fig. 7.—Flow curves of an extracorporeal circuit with gravity venous discharge and roter pump. *A*, Arterial return—true flow curve measured with the orifice-plate flowmeter. *B*, Arterial return—mean flow measured with the orifice plate flowmeter. *C*, Gravity venous discharge—measured with the gravity discharge meter. *D*, Integrated total flow curve.

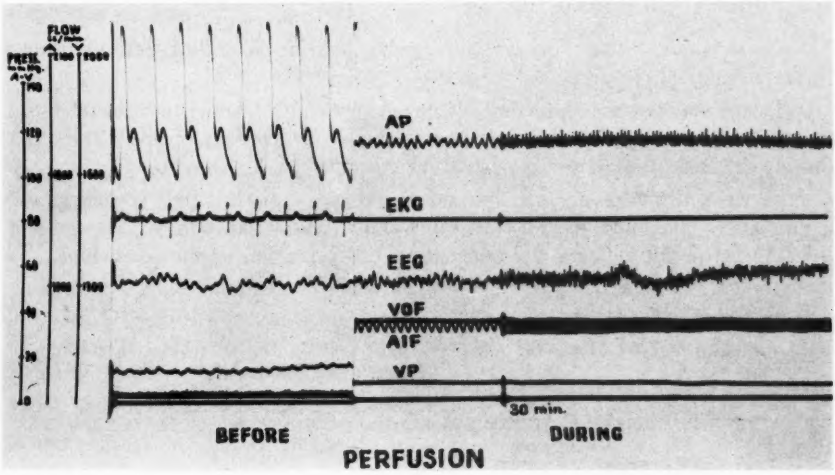

Fig. 8.—Monitoring curves of a cardiopulmonary bypass: *AP*, arterial (perfusion) pressure; *VP*, central venous pressure; *AIF*, arterial (return) inflow; *VOF*, venous (gravity) outflow.

We use this flowmeter, as a direct continuation of the venous outflow tubing, mounted on the venous reservoir. Its circulatory resistance is equal to that of a plastic tubing, 3/8 inch inner diameter and 1 inch in length; it does not interfere with the function of the pump; it does not impede the venous outflow; and it is not a source of any detectable hemolysis.

This simple device could be used for direct flow determinations even without the secondary elements; however, from the standpoint of distant recording and undisturbed observation, we found the first mentioned secondary element very useful. The tertiary device, which consists of the photoelectric cells and the special signal system, may open further possibilities in the automatization of the artificial heart-lung machine.

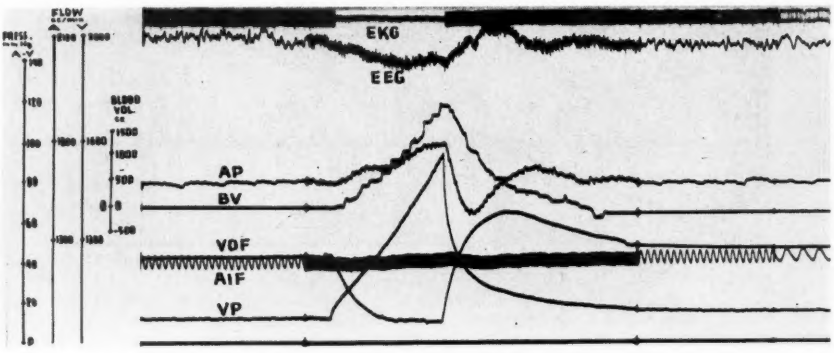

Fig. 9.—Monitoring curves of a total body perfusion. Effect of the temporary constriction of the venous outflow tubing. *BV*, Blood volume.

SUMMARY AND CONCLUSIONS

The authors discuss briefly the significance of blood-flow determinations in extracorporeal circuits.

Two flowmeters are described. One is especially designed to measure blood flow through the arterial (return) line of the extracorporeal circuit. The other flowmeter is useful to determine venous gravity outflow.

In clinical practice both flowmeters proved to be simple, accurate, and atraumatic. Their use increases safety in open-heart operations and opens new possibilities in physiologic observations in the perfusion of human beings.

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MASSIVE HEMOLYSIS IN EXTRACORPOREAL CIRCULATION

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MASSIVE HEMOLYSIS has been feared as a complication of operations involving an extracorporeal pump-oxygenator for cardiopulmonary bypass but this complication has not been reported. After successful use of the De Wall-Lillehei pump-oxygenator in 26 patients at this center, massive hemolysis was encountered in the next 2 patients. The purpose of this communication is to record these instances of excessive hemolysis and to report the nature and results of laboratory efforts to determine the cause of this complication.

CASE REPORTS

CASE 1.—D. L. M. A 12-year-old white boy, weighing 35 kilograms, with aortic stenosis underwent open-heart correction with the use of cardiopulmonary bypass on Aug. 5, 1959. The duration of bypass was 12 minutes with a perfusion rate of 2,500 c.c. per minute. Immediately following completion of the operation, the patient was noted to have gross hemoglobinuria. Serum hemoglobin at that time was 1,400 mg. per cent. Consultation was obtained from the medical service and Solu-Cortef was administered for approximately 24 hours. In addition, 4 per cent urea (1, 2, 3, 4) and M/6 sodium lactate were administered intravenously at a rate of 3 c.c. per minute to obtain diuresis and alkalinization of the urine. Within 24 hours the urine was clear and the remainder of the hospital course was uneventful.

CASE 2.—E. M. L. A 5-year-old white girl, weighing 30 kilograms, with pulmonary valvar stenosis and patent ductus arteriosus underwent open-heart correction of pulmonary valvar stenosis and division and suture of the patent ductus arteriosus using cardiopulmonary bypass with a bubble oxygenator (Aug. 12, 1959). The patient was on bypass for 30 minutes with a perfusion rate of 2,000 c.c. per minute. Following completion of the surgery the patient was noted to have hemoglobinuria and plasma hemoglobin of 2,000 mg. per cent. Medical consultation was obtained and exchange transfusion was done through the right saphenous vein with approximately 2,000 c.c. of whole fresh blood. The patient was given Solu-Cortef, and 4 per cent urea and M/6 sodium lactate were administered intravenously to promote diuresis and alkalinization. After approximately 24 hours the urine had cleared and the patient recovered satisfactorily.

CASE 3.—S. K. H. A 17-year-old white girl, weighing 45 kilograms, with a ventricular septal defect had this defect closed on Aug. 19, 1959, using cardiopulmonary bypass with a bubble oxygenator with only one change from the previous 2 cases in that large helix tubing

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was used in place of small helix tubing. The patient was on the bypass 20 minutes with a perfusion rate of 2,860 c.c. per minute. Following completion of the operation, the patient's plasma hemoglobin was 58 mg. per cent. There was no evidence of hemoglobinuria.

LABORATORY STUDIES

Following each instance of severe hemolysis, consideration was given to possible causes of this alarming occurrence. The surgical and technical personnel of the operating team had not changed and there had been no recognized change in surgical technique. The pump-oxygenator used was a bubble oxygenator as described by De Wall and his co-workers[5] and was not changed from that used in previous successful cases. The flow rates and duration of bypass were within the limits previously used without hemolysis.

The possibility of transfusion reaction was investigated by rematching all of the blood used and no incompatibility was found. An hemantigen test was made on both donor and recipient's blood but no abnormal antigen was present. These results appear to prove that transfusion reaction was not responsible for the observed hemolysis.

In the laboratory the pump was assembled exactly as it was in surgery and, using fresh heparinized dog blood, within one minute after starting the pump grossly noticeable hemolysis was present. The pump was adjusted to make it barely occlusive. The fingers of the pump were working correctly and it was not felt that pump trauma could be the cause of the hemolysis. A different Sigmamotor pump was used as a check with the same results.

The process of cleaning tubing was carefully checked, as a known hemolytic agent, Alconox, had been used. However, hemolysis resulted whether the tubing was cleaned with Alconox, with plain water, or even if it was not cleaned prior to use. Osmolarity changes during the run were checked with Fiske osmometer and there were no significant changes. All metallic parts were resiliconized and again the hemolysis occurred. It was felt that the remaining probable source of hemolysis was the plastic tubing. Pieces of tubing approximately one foot in length were filled with 50 c.c. of whole, fresh, heparinized dog blood and incubated in a water bath at 38° C. Grossly evident hemolysis occurred in the small helix tubing after 15 minutes and there was no gross hemolysis in the other sizes of tubing. Plasma hemoglobins were not obtained during this part of the experiment. The bubble oxygenator was again assembled as in the previous experiment and when the small helix tubing was bypassed no gross hemolysis was apparent. When the small helix was incorporated hemolysis began to appear.

Two bubble oxygenators were then set up using a segment of the small helix tubing in question in one and a different piece of large helix tubing in a second. Fresh heparinized dog blood was then drawn and one half of the blood was used in the small helix pump-oxygenator and the other half of the blood from the same dog in the large helix pump-oxygenator. At flows of 1,140 c.c. per minute for 60 minutes, there was a rather marked difference in the amount

of hemolysis with the large helix as compared to the small helix (Table I). The small helix had at least 5 times as great amount of hemolysis as the large helix after identical flow volumes.

TABLE I. PUMPING THROUGH LARGE AND SMALL HELIX

	BLOOD FLOW (1,140 C.C./MIN.)							
	TIME (MIN.)							
	1	5	10	15	20	25	30	60
Large helix	0*	0			3	5	9	27
Small helix	2	5	12	33	36	75	86	165

*Values reported in mg. % of plasma hemoglobin.

DISCUSSION

Hemolysis of blood in the small helix tubing was successively reproduced but with serial tests of the small helix tubing there was progressively less hemolysis evidenced, both grossly and by plasma hemoglobin determinations. It was believed on this basis that the agent in the tubing causing the hemolysis was slowly deactivated or dissipated. Hemolysis had not been a problem prior to these 2 cases and Case 3 demonstrates that after changing only the helix tubing in our setup that massive hemolysis was not obtained. Subsequently 36 patients have been operated upon employing the bubble oxygenator (3 patients) or the rotating disc oxygenator[6] (33 patients) without significant hemolysis. The tubing used in the two clinical cases demonstrating massive hemolysis and the tubing used in the laboratory were all from a single 60 foot piece of helix tubing and the hemolysis could not be demonstrated from other lots of small helix tubing. We were unable to identify the actual hemolytic factor in the tubing. In reviewing the literature, no previous reports of hemolysis from this cause were found and this is submitted as a new cause of massive hemolysis and a rare complication of extracorporeal circulation.

Since the substance causing hemolysis appeared to be unstable, becoming less evident with each subsequent exposure to blood in the laboratory, it is suggested that batches of new tubing be tested with blood to observe hemolysis.

A detailed discussion of the treatment of massive hemolysis is beyond the scope of this communication. In these patients a regimen consisting of rapid alkalinization, early administration of cortisone, osmotic diuresis using urea, and exchange transfusion have been effective.

CONCLUSIONS

1. Massive hemolysis following cardiopulmonary bypass has been encountered in 2 consecutive patients.

2. Laboratory investigation indicates the presence of an unstable hemolytic agent in a single segment of plastic tubing.

3. This experience suggests the need for routine testing of new segments of plastic tubing.

4. Treatment of these patients with cortisone, sodium lactate, urea, and exchange transfusion has been effective.

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ABERRANT RIGHT SUBCLAVIAN ARTERY WITH KOMMERELL'S DIVERTICULUM

Report of a Case

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THE ANOMALOUS origin of the right subclavian artery from the descending aorta has long been recognized and the first case was reported by Hunauld in 1735, according to Holzapfel.[1] It has only been since Gross's original article[2] in 1946 on the division of this vessel that the diagnosis has had therapeutic significance. As reported by Gross, and numerous others, simple division of this vessel for the relief of dysphagia lusoria is technically easy and therapeutically satisfactory. In a recent case, technical difficulties were encountered related to an uncommon variation of this anomaly, namely the presence of so-called Kommerell's diverticulum. The first detailed description of this anomaly was published by Kommerell in 1936,[3] and his explanation, as outlined in the accompanying diagram (Fig. 1), has generally been accepted. Actually it is not a diverticulum, but, as he points out, simply persistence of the distal end of the right aortic arch, and, indeed, was seen much earlier and reported in the extensive review of this anomaly by Holzapfel,[1] who mentions a swelling of the aortic end of this vessel in several instances. The ante-mortem diagnosis of Kommerell's diverticulum was first made roentgenologically by Stauffer and Pote,[4] who saw what they called an aneurysmal dilatation above the aortic arch and raised the question of whether or not this was a diverticulum, although the patient was not operated upon. These same, rather typcial x-ray findings were noted in the present case but they were not correctly interpreted preoperatively.

It seemed worth while to report the technical difficulties associated with such a situation if it is encountered unexpectedly in the division of an aberrant right subclavian artery. Moreover, awareness of this possibility might prevent exploration of a lesion which, by x-ray examination, appears to be a posterior mediastinal tumor.

CASE REPORT

(Salem Hospital—No. A33290.) The patient was a 62-year-old widow who entered the hospital with a history of recurrent attacks of upper substernal and back pain between the shoulder blades during the previous 5 years. The attacks had become much more

From the Salem Hospital, Salem, Mass.
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frequent in the months just before admission. Recently the pain seemed to be associated with lying down, and often lasted as long as half an hour. Nitroglycerine did not relieve the pain but sitting up gave prompt relief. Dysphagia had never been noted by the patient. Extensive studies previously had failed to reveal any abnormality in the lungs or cardiovascular system. Fluoroscopy just prior to this admission did reveal a superior mediastinal mass.

As noted in Figs. 2 and 3, barium swallow disclosed a left superior mediastinal mass with some distortion of the esophagus, with the possibility of an anomalous subclavian artery. The mass itself was considered preoperatively to be extra-aortic, and it apparently did not pulsate.

Physical examination and laboratory studies were entirely within normal limits and the patient was explored with the preoperative diagnosis of probable mediastinal tumor, although the possibility of an anomalous subclavian artery was definitely entertained.

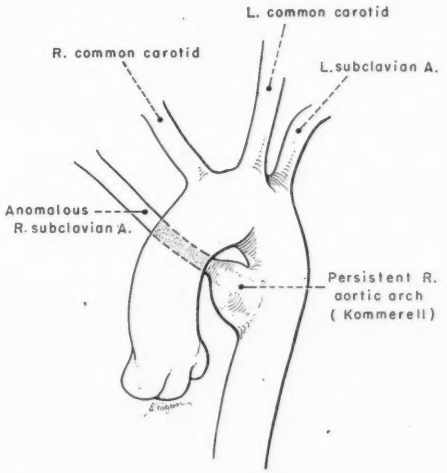
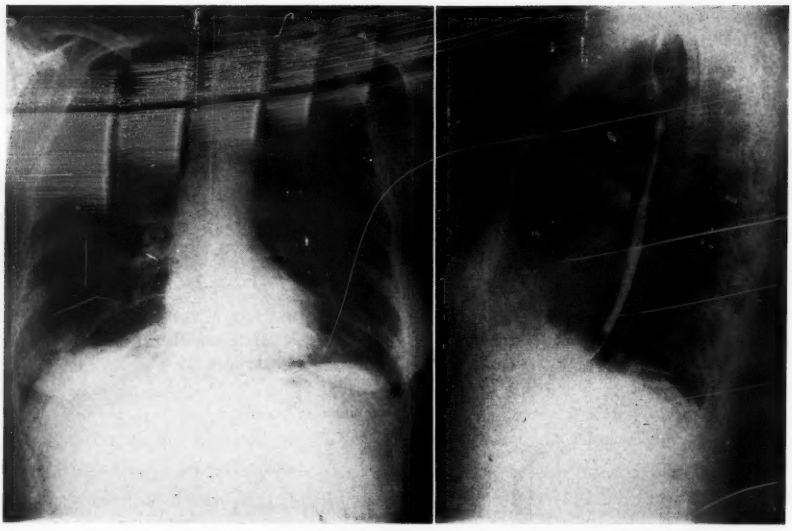


Fig. 1.—Illustration of Kommerell's diverticulum which is actually persistence of the right aortic arch from which the anomalous right subclavian artery arises.

A standard left posterolateral thoracotomy was used, giving excellent exposure of what proved to be an anomalous right subclavian artery, arising just distal to the left subclavian artery, from the posterior medial aspect of the aortic arch. At the aortic end there was a definite swelling, which was believed to be aneurysmal, although in retrospect it was undoubtedly a Kommerell's diverticulum. The subclavian artery was freed distal to this swelling, and divided, following which the presumed aneurysm was resected. Technically this was complicated by the fact that there was no neck to the abnormal swelling and the aortic arch in this area was very atherosclerotic. Considerable bleeding occurred before closure was finally accomplished with two running layers of 2-0 cotton sutures. The suture line was considered quite precarious, but no leakage occurred and the chest was closed in a routine manner.

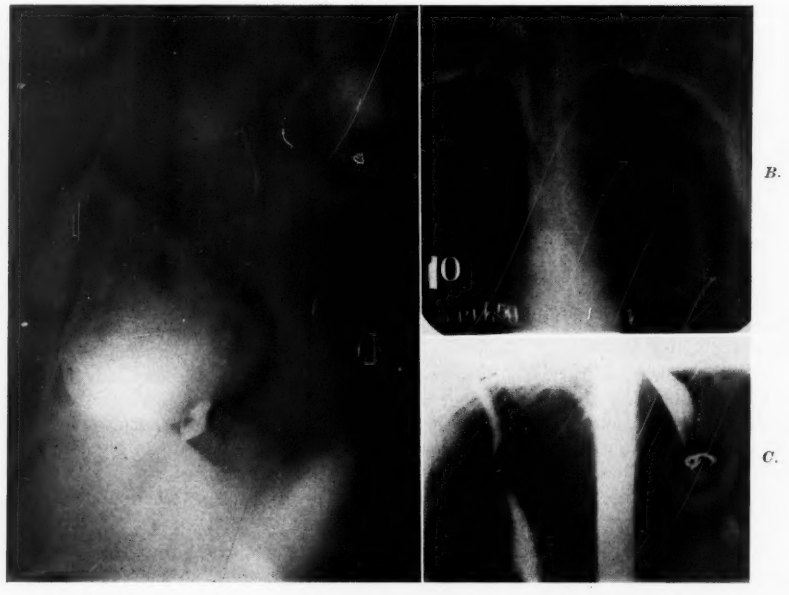
Postoperatively, the patient did quite well and was discharged in 10 days in good condition. Subsequent examinations revealed no abnormalities, although the patient did develop a little hoarseness, which was felt to be due to partial paralysis of the left recurrent laryngeal nerve. Three months postoperatively, however, she suddenly developed a severe cough and, while sitting in the office of her cardiologist, suddenly began to cough up gross blood and was pronounced dead on arrival at the hospital about 20 minutes later.



A. *B.*

Fig. 2.—*A*, Posteroanterior view of the chest which shows a calcified mass superior and medial to the arch of the aorta, causing slight deformity of barium in the esophagus.

B, Lateral film which shows posterior indentation of the barium-filled esophagus immediately above the aortic arch.



A. *B.* *C.*

Fig. 3.—*A*, Barium-outlined esophagus (retouched in left anterior oblique projection) which reveals left posterolateral impression on esophagus.

B, Laminagraph sections, exposed 10 cm. above the table top, which reveal triangular band of soft tissue density extending obliquely cephalad and to the right immediately above and adjacent to the aortic arch.

C, Left anterior oblique spot film of barium-filled esophagus again revealing left posterolateral encroachment on the esophagus.

Postmortem examination disclosed that the suture line of the aortic arch had ruptured into the apex of the left lung, where a large abscess cavity was found. The patient had hemorrhaged via the bronchial tree.

DISCUSSION

The unfortunate outcome of this case has pointed up several ways in which such difficulties might well be avoided. Obviously a correct preoperative diagnosis would be of great assistance, and aortography would certainly outline this lesion in detail in most instances. As noted in the present case, and in at least one other, the diagnosis can be made by routine x-ray examination. If this anomaly is recognized preoperatively, it might well be wiser simply to divide the subclavian vessel where it is of normal size, as it is unlikely that this abnormal persistence of the right aortic arch would rupture or thrombose in such a way as to lead to future complications. If resection were felt to be safer, certain measures could be taken to reinforce the aortic suture line, such as encircling the aorta in this area with a cuff of Teflon, since there will be no narrow neck. By excising the entire diverticulum one is actually left with a defect in the aortic wall, which must be closed with sutures under some tension. Conceivably a patch of synthetic material could be used to close such a defect, but this would probably entail complete isolation of the aortic circulation, which hardly seems justified for a lesion that can be handled more easily in another fashion.

SUMMARY

A case of resection of an anomalous right subclavian artery with Kommerell's diverticulum and the diagnosis and therapeutic complications of its therapy discussed.

The author would like to acknowledge the assistance of Dr. Henry D. Stebbins, who originally referred the patient to me, and Dr. Robert Liming, who interpreted the x-rays.

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3. Kommerell, B.: Verlagerung des Oesophagus durch eine abnorm verlaufende Arteria subclavia dextra (Arteria lusoria), Fortschr. a. d. Geb. d. Röntgenstrahlen **54**: 590-595, 1936.
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A NEW TYPE SUCTION TIP ESPECIALLY DESIGNED FOR INTRACARDIAC SURGERY

Frederick S. Cross, M.D., and Richard D. Jones, B.A.,
Cleveland, Ohio

THE DEVELOPMENT of new surgical procedures often necessitates instruments of new design. To do direct vision open intracardiac surgery, it is essential that the surgeon work in a dry operative field. This can be accomplished by means of coronary artery inflow stasis, with or without elective cardiac arrest, or by means of a suitable sump-type suction tip properly placed to remove the coronary return flow from the beating heart. Although the ordinary surgical suction tips are available, they have been only partially adaptable to the needs of open-heart surgery in our experience. The suction tip to be described has been used for the past 4 years, both in the laboratory and clinically, and has been found to be exceedingly satisfactory.

DESCRIPTION

The major feature of the suction tip is a series of radial fins or flutes, 14 to 18 in number, which are an integral part of the tube comprising the tip (Fig. 1). These flutes function to keep tissue away from the suction holes as does the outer jacket on the usual sump-type suction tip. The tip is constructed by drilling a hole through the axis of a length of drawn brass pinion wire to form a tube. A cross section of this tube has the shape of a gear wheel. Each tooth of the gear represents a flute which extends the length of the tube, projecting from the wall to form a series of longitudinal grooves. The tube is open at the distal end, and pierced openings are placed at the bottom of each groove to form a ring of holes 1 mm. from the end of the tube. The proximal end of the suction tip is threaded to fit handles of conventional design. The tip is then polished and chrome plated. When used to return blood to the oxygenator during open-heart surgery, the tips and handles are coated with silicone resin (No. 803),* baked, and lightly coated with Antifoam A.*

Three sizes are in use. The smallest is 2.7 cm. long, with an outside diameter of 6 mm., and an inside diameter of 2.5 mm. It is most useful where

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*Dow Corning Corporation, Midland, Mich.

exposure is limited, as in small children, or within the aorta during aortic valve exposure. The medium-sized tip is 5.6 cm. long, with an outside diameter of 7.9 mm., and a 3 mm. bore. The largest tip is the same length, with an outside diameter of 15 mm., and a possible internal diameter of 6.4 mm. The groove depth varies from 0.9 mm. to 1.2 mm., and the width of the groove 0.9

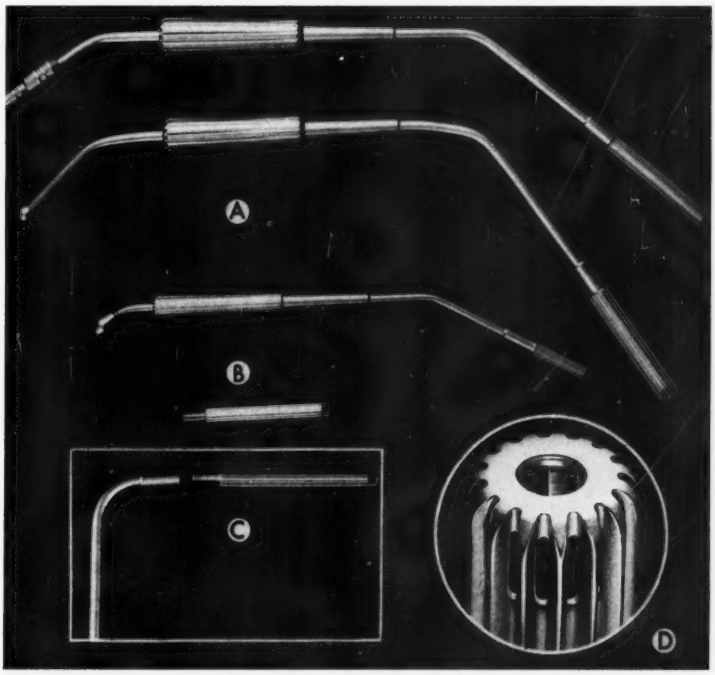

Fig. 1.—*A*, Photograph of suction tips used most frequently at present. *B*, Medium-sized tip with handle removed. *C*, Right angle handle design suggested by Dr. Herbert Sloan of the University of Michigan. *D*, Close-up of suction tip to show details of construction at distal end.

mm. to 1.4 mm. The long diameter of the circumferential oval openings at the distal end of the tip range from 1.9 mm. in the small tip to 4.0 mm. in the large one. The larger bore tips are partially closed at the distal end to limit that opening to 4 mm.

DISCUSSION

The suction tip was designed as a sump-type suction to handle large blood flows. It functions efficiently in deep pools of blood encroached upon by surrounding tissues, as well as in shallow pools where a surface action is needed. It remains free from plugging, and is easily cleaned and sterilized. The tips are readily interchangeable in the event that it becomes desirable. The principal advantage of the tip design is that suction is applied directly to the

surface of tissues in such a manner that the tissue is not drawn across the openings to occlude them. This is accomplished by the closely spaced longitudinal flutes which support the tissue away from the openings, and by the grooves between the flutes which always communicate with a source of air or fluid. This is true even though the tip may be thrust deeply into tissue pockets.

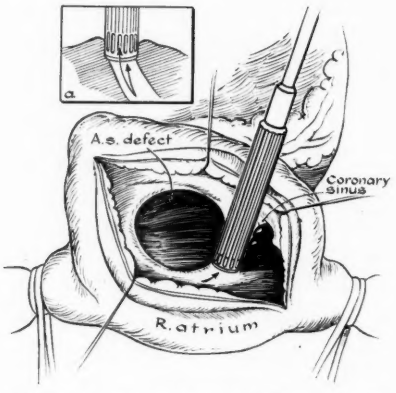

Fig. 2.—Drawing to illustrate "vacuum cleaner" action of suction tip in removing shallow pools of blood. Inset shows tip placed firmly in coronary sinus opening to handle this source of blood contamination.

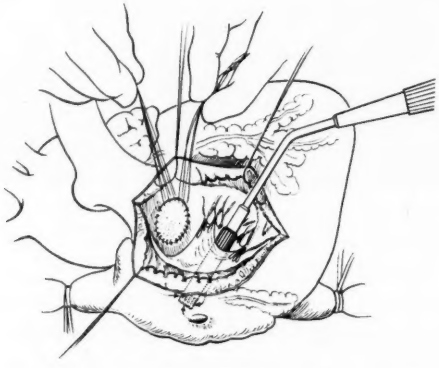

Fig. 3.—In the repair of ventricular septal defects, the fluted design of the tip allows it to function effectively in deep pools of blood within the heart.

Injury to tissues is minimized because large suction forces are not applied directly to the tissues enveloping the tip as occurs when an unshielded tip is occluded by tissue.

Special attention was given to the design of the suction probe so that the maximum suction is at the extreme end of the tip. This simulates a "vacuum cleaner" effect in the removal of very shallow pools of blood to give an operative field that is virtually dry (Fig. 2). Such action is especially

important in the repair of atrial septal defects where the defect is in a dependent position. The thin layer of blood over the atrial septum can be removed gently, leaving the blood level at the rim of the defect. It is possible to place the suction tip snugly into the coronary sinus opening to handle effectively this source of blood contamination of the operative field in a manner superior to other suction tips that we have tried. The flat end of the tip with its single opening, as well as the terminally placed circumferential openings between protecting flutes, afford this action. The tip has proved equally effective in blood removal in the repair of ventricular septal defects, or in the correction of tetralogy of Fallot, during which it is placed deeply within the heart through the tricuspid valve into the right atrium. Papillary muscles, delicate valve leaflets, and chordae tendineae are not traumatized by suction against the tip openings even with large suction flows (Fig. 3). Because the tip is designed without an outer jacket, it can be made with a large internal diameter and a relatively small outside diameter to reduce the bulk. The large internal diameter allows large flows with less suction force and less resulting trauma to the blood which is returned to the oxygenator for recirculation.

Although these suction probes were designed for use in open intracardiac surgery, they have been valuable in all types of cardiovascular and thoracic surgery, and certainly would be of equal value in general surgical procedures.

SUMMARY

1. An intracardiac suction tip of new design is described.*
2. The uses and advantages of this tip are discussed.

*Manufactured by Pemco, Inc., 5663 Brecksville Road, Cleveland 31, Ohio.

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Forty-First Annual Meeting
April 24, 25, 26, 1961
Sheraton Hotel, Philadelphia, Pa.

Program

MEMORANDA FOR GUIDANCE OF SPEAKERS

1. In accordance with the By-Laws of the Association, the papers which are read at the meeting shall be given to the Reporter *immediately after presentation.* If these papers are not submitted promptly their publication may be delayed.
2. In publication it is customary to group discussions together on a series of papers. Any delay in the return of corrected discussions to the Editor means that publication of all papers on the subject will be held up. This is manifestly unfair to those who are conscientious in the prompt submission of their remarks for publication. Undue tardiness may mean that the discussion will not be published.
3. *Regular Program:* Speakers will be limited to 15 minutes.
4. *Thoracic Surgery Forum:* Speakers will be limited to 10 minutes. Papers on the Thoracic Surgery Forum as submitted for publication will be limited to 3,000 words. At the discretion of the President, limited discussion will be allowed the following formal presentations.
5. *Discussion of Papers:* Discussers will be limited to 3 minutes. Only members of the Association and invited speakers will have the privilege of discussing papers.

Monday Morning, April 24, 1961

8:30 A.M. Business Session (Limited to Members)
Grand Ballroom

8:45 A.M. Scientific Session: REGULAR PROGRAM
Grand Ballroom

1. **Traumatic Tracheal Rupture.** Robert R. Shaw, Donald L. Paulson, and John L. Kee, Jr., Dallas, Texas.
2. **The Surgical Management of Metastatic Neoplasms in the Lung.** Earle W. Wilkins, Jr., John F. Burke (by invitation), and John M. Head (by invitation), Boston, Mass.
3. **Bronchiolar Cell Carcinoma of the Lung: A Review of 33 Patients.** Hugh F. Fitzpatrick, Robert E. Miller (by invitation), Malcolm S. Edgar, Jr. (by invitation), and Charles F. Begg (by invitation), New York, N. Y.
4. **Routine Use of the Carlens Double-Lumen Endobronchial Catheter: An Experimental and Clinical Study.** Robert W. Newman, George E. Finer (by invitation), and James E. Downs (by invitation), Knoxville, Tenn.

5. **Complete Functional Restitution of the Food Passage in Extensive Stenosing Caustic Burns.** Joseph H. Ogura (by invitation), Charles L. Roper (by invitation), and Thomas H. Burford, St. Louis, Mo.
6. **Incompetence of the Gastric Cardia Without Radiological Evidence of Hiatus Hernia.** Clement A. Hiebert (by invitation), and Ronald Belsey (by invitation), Bristol, England.

Monday Afternoon, April 24, 1961

2:00 P.M. Scientific Session: REGULAR PROGRAM
Grand Ballroom

7. **Tumors of the Thymus Gland.** Philip E. Bernatz, O. Theron Clagett, and Edgar G. Harrison, Jr. (by invitation), Rochester, Minn.
8. **The Etiology of Acute Respiratory Acidosis.** Richard M. Peters, Thomas B. Barnett (by invitation), and Robert Zeppa (by invitation), Chapel Hill, N. C.
9. **Perfusion Hypothermia and Ventricular Fibrillation.** William J. Kerth (by invitation), John J. Osborn (by invitation), Frank Gerbode, J. Bruce Johnston (by invitation), and Takeshi Ogata (by invitation), San Francisco, Calif.
10. **Alterations in Blood Volume Following "Normovolemic" Total Body Perfusion.** Robert S. Litwak (by invitation), Albert J. Gilson (by invitation), Ralph J. Slonim (by invitation), Caspar C. McCune (by invitation), and Howard L. Gadboys (by invitation), Coral Gables, Fla.
11. **A Study of the Causes of 60 Deaths Following Total Cardiopulmonary Bypass.** Jean P. Despres (by invitation), Riccardo Benvenuto (by invitation), and John C. Callaghan, Edmonton, Canada.
12. **Coordinated Postsystolic Myocardial Augmentation Combined With Systolic Neutralization: Development and Clinical Application to the Failing Heart.** David H. Watkins, E. R. Duchesne (by invitation), and Byron E. Pollock (by invitation), Denver, Colo.
13. **Clinical Results in Open Mitral Valvuloplasty.** Joe D. Morris and Herbert Sloan, Ann Arbor, Mich.

Tuesday Morning, April 25, 1961

8:30 A.M. Scientific Session: THORACIC SURGERY FORUM
Grand Ballroom

14. **Profound Hypothermia With Simplified Equipment: A Disposable Stainless Steel Blood Heat Exchanger of High Efficiency.** William G. Esmond (by invitation), John Stram (by invitation), Safuh Attar (by invitation), and R Adams Cowley, Baltimore, Md.
15. **Gas Chromatography: A Simple, Rapid, Reliable Method for Blood Gas Analysis.** Russell H. Wilson (by invitation), Bruce Jay (by invitation), and Robert H. Holland, Dallas, Texas.
16. **A New Reflection Oximeter.** P. F. Ware, M. L. Polanyi (by invitation), R. M. Hehir (by invitation), J. F. Stapleton (by invitation), J. I. Sanders (by invitation), and S. L. Kocot (by invitation), Worcester, Mass.
17. **The Repair of Circumferential Defects of the Trachea by Direct Anastomosis: Experimental Evaluation.** James R. Cantrell (by invitation), Seattle, Wash., and J. Roland Folse (by invitation), Bethesda, Md.
18. **The Pulmonary Arterial Blood Flow Through an Acutely Atelectatic Lung.** Rudolph C. Camishion (by invitation), Yoshinori Ota (by invitation), Vincent D. Cuddy (by invitation), and John H. Gibbon, Jr., Philadelphia, Pa.

19. **The Effects of Positive Pressure Lung Inflation upon Pulmonary Vascular Dynamics.** Paul H. Gerst (by invitation), New York, N. Y.
20. **Bronchogenic Carcinoma Produced Experimentally in the Dog.** E. J. Beattie, Jr., E. W. Staub (by invitation), N. O. Correll (by invitation), and G. Hass (by invitation), Chicago, Ill.
21. **Hematoporphyrin Derivative: A New Aid for the Endoscopic Detection of Malignancy.** Richard L. Lipson (by invitation), Edward J. Baldes (by invitation), and Arthur M. Olsen, Rochester, Minn.
22. **Lung Resection With Temporary Vascular and Bronchial Occlusion.** F. John Lewis, Nicholas J. Demos (by invitation), Peter J. Connaughton (by invitation), and Stuart Poticha (by invitation), Chicago, Ill.
23. **Modified Non-Suture Anastomosis of Coronary and Internal Mammary Arteries in Dogs.** George J. Magovern (by invitation), Edward M. Kent, Bernard S. Levowitz (by invitation), Ram S. Ratan (by invitation), John B. Lovette (by invitation), and Sheldon O. Burman (by invitation), Pittsburgh, Pa.
24. **Effects of Continuous Flow Through Implanted Mammary Artery and Myocardial Ischemia on Mammary-Coronary Coommunications.** Mariano Lopez-Belio (by invitation), Luis Sanchez (by invitation), Salvador Rodriguez (by invitation), and Ormand C. Julian, Chicago, Ill.
25. **Surgical Correction of Coronary Arteriovenous Fistula.** Osler A. Abbott, Carlos Rivarola (by invitation) and R. Bruce Logue (by invitation), Atlanta, Ga.
26. **Total Mitral Valve Replacement: The Shielded Ball Valve Prosthesis.** Albert Starr (by invitation), and M. Lowell Edwards (by invitation), Portland, Ore.
27. **Prolonged Survival After Total Replacement of the Mitral Valve in Dogs.** Ara V. Doumanian (by invitation), and F. Henry Ellis, Jr., Rochester, Minn.
28. **Total Excision of the Mitral Valve and Replacement With the Autologous Pulmonic Valve.** Richard R. Lower (by invitation), Raymond C. Stofer (by invitation), and Norman E. Shumway (by invitation), San Francisco, Calif. (Sponsored by Emile Holman, San Francisco, Calif.)

Tuesday Afternoon, April 25, 1961

2:00 P.M. Executive Session (Limited to Active and Senior Members)
Grand Ballroom

3:00 P.M. Scientific Session: REGULAR PROGRAM
Grand Ballroom

Address by the President, John H. Gibbon, Jr., Philadelphia, Pa.
Address by Honored Guest, Professor A. L. d'Abreu, O.B.E., M.B., Ch.M., F.R.C.S. Dean of the Faculty of Medicine, University of Birmingham, England. **Thoracic Surgery in the Commonwealth of Medicine.**

29. **Surgical Considerations in Occlusive Disease of the Great Vessels Arising From the Aortic Arch.** E. Stanley Crawford, Michael E. De Bakey, and George C. Morris, Jr. (by invitation), Houston, Texas.
30. **Coarctation of the Aorta: A Review of 500 Cases.** Samuel R. Schuster (by invitation), and Robert E. Gross, Boston, Mass.

7:00 P.M. Banquet and Dancing, Grand Ballroom.
Attendance limited to Members of the Association and their ladies, Invited Authors and Co-authors and their ladies.
Dinner dress preferred.

Wednesday Morning, April 26, 1961

9:00 A.M. Scientific Session: REGULAR PROGRAM
Grand Ballroom

31. **The Anatomy and Embryology of Endocardial Cushion Defects.** L. H. S. Van Mierop (by invitation), Ralph D. Alley, Harvey W. Kausel, and Allan Stranahan, Albany, N. Y.
32. **The Surgical Management of Complete Common Atrioventricular Canal.** James V. Maloney, Jr., Samuel A. Marable (by invitation), and Donald G. Mulder (by invitation), Los Angeles, Calif.
33. **Surgical Considerations for Treatment of Congenital Tricuspid Atresia and Stenosis.** Raymond K. Bopp (by invitation), Parry B. Larsen (by invitation), Joan L. Caddell (by invitation), James R. Patrick (by invitation), and William W. L. Glenn, New Haven, Conn.
34. **Surgical Correction of Congenital Supravalvular and Valvular Aortic Stenosis Using Deep Hypothermia and Circulatory Arrest.** Archer S. Gordon (by invitation), Bertrand W. Meyer, and John C. Jones, Los Angeles, Calif.
35. **An Improved Transatrial Approach to the Closure of Ventricular Septal Defects.** Allen S. Hudspeth (by invitation), A. Robert Cordell (by invitation), Jesse H. Meredith (by invitation), and Frank R. Johnston, Winston-Salem, N. C.
36. **Aortic-Cardiac Fistulas Following Corrective Operations for Ventricular Septal Defect and Tetralogy of Fallot.** M. Weinberg, Jr., M. H. Agustsson (by invitation), B. M. Gasul (by invitation), E. H. Fell, J. P. Bicoff (by invitation), Z. Steiger (by invitation), T. Iwa (by invitation), and R. Arcilla (by invitation), Chicago, Ill.
37. **Cardiac Surgery in the Newborn.** John L. Ochsner (by invitation), and Denton A. Cooley, Houston, Texas.

Wednesday Afternoon, April 26, 1961

2:00 P.M. Scientific Session: THORACIC SURGERY FORUM
Grand Ballroom

38. **Parenchymatous Splenopulmonary Anastomosis as Possible Treatment for Portal Hypertension in Children.** Francis Remillard (by invitation), Patrick E. Conen (by invitation), Toronto, Ont., and George R. Walker, Sudbury, Ont.
39. **Alterations in Intrabronchial Temperature, Humidity, and Oxygen Concentration Produced by Various Clinical Methods of Oxygen Administration.** JD Mortensen (by invitation), Salt Lake City, Utah.
40. **The Regression of Pulmonary Vascular Disease After the Creation of Pulmonary Stenosis.** J. Francis Dammann, Jr., Charlottesville, Va., James A. McEachen (by invitation), Santa Monica, Calif., W. M. Thompson, Jr. (by invitation), Charlottesville, Va., Rodney Smith (by invitation), Santa Monica, Calif., and William H. Muller, Jr., Charlottesville, Va.
41. **The Effects of Low Molecular Weight Dextran Upon the Blood Flow Rate During Extracorporeal Circulation.** Christopher Drake (by invitation), Fidel Macalalad (by invitation), and F. John Lewis, Chicago, Ill.
42. **Mechanisms of Pulmonary Hypertension in Acute Hypoxia.** Ibrahim K. Dagher (by invitation), Henry G. Mishalany (by invitation), Beirut, Lebanon, and F. A. Simeone (by invitation), Cleveland, Ohio. (Sponsored by John L. Wilson, Beirut, Lebanon.)

43. **Experiences With NaI^{131} Injected Into the Myocardium as an Estimate of Coronary Blood Flow.** Irving M. Madoff, and William Hollander (by invitation), Boston, Mass.

44. **Replacement of Right Ventricular Myocardium With a Teflon Prosthesis.** Harold A. Collins (by invitation), J. Kenneth Jacobs (by invitation), Robert T. Sessions (by invitation), and Rollin A. Daniel, Jr., Nashville, Tenn.

45. **Blood-Brain Barrier Studies in Extracorporeal Cooling and Warming.** Harry S. Pollard, Jr. (by invitation), R. J. Fleischaker (by invitation), J. J. Timmes, and K. E. Karlson, St. Albans, N. Y.

46. **Hemodynamic and Metabolic Responses of the Whole Body and Individual Organs to Cardiopulmonary Bypass With Profound Hypothermia.** Thomas J. Yeh (by invitation), Lois T. Ellison (by invitation), and Robert G. Ellison, Augusta, Ga.

47. **Metabolic Alterations Associated With Profound Hypothermia and Extracorporeal Circulation in the Dog and Man.** William F. Bernhard (by invitation), Hans F. Schwarz (by invitation), and Robert E. Gross, Boston, Mass.

48. **Treatment of Respiratory Insufficiency by Prolonged Extracorporeal Circulation: Experimental Observations.** Robert Schramel, William Chapman (by invitation), Berwin Volnie (by invitation), and Oscar Creech, Jr., New Orleans, La.

49. **Correction of Complete Heart Block by a Self-Contained and Subcutaneously Implanted Pacemaker.** William M. Chardack (by invitation), Andrew A. Gage (by invitation), and Wilson Greatbatch (by invitation), Buffalo, N. Y.